Inventor search history

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=> d his L74

     (FILE 'HCAPLUS' ENTERED AT 13:59:36 ON 28 NOV 2007)
                SAVE TEMP L73 BET232HCTX/A
                E BOLDT M?/AU
L74           6 S E2,E10

=> d que L74
L74           6 SEA FILE=HCAPLUS ABB=ON  PLU=ON  ("BOLDT M"/AU OR "BOLDT
                MATTHIAS"/AU)


=> d his L90

     (FILE 'MEDLINE, BIOSIS, EMBASE, DRUGU' ENTERED AT 14:52:16 ON 28 NOV 2007)
L90          19 S L89 AND L72
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=> d que L90
L72              QUE  ABB=ON  PLU=ON  AY<2004 OR PY<2004 OR PRY<2004 OR RE
                 VIEW/DT
L74           6 SEA FILE=HCAPLUS ABB=ON  PLU=ON  ("BOLDT M"/AU OR "BOLDT
                MATTHIAS"/AU)
L86          49 SEA L74
L89          20 SEA L86 AND (ADMINIST? OR TREAT? OR SUPPLEM? OR SPORT? OR
                PERFORM? OR THERAP? OR PHARMAC?)
L90          19 SEA L89 AND L72


=> dup rem L74 L90
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PROCESSING COMPLETED FOR L90
L91          16 DUP REM L74 L90 (9 DUPLICATES REMOVED)
                ANSWERS '1-6' FROM FILE HCAPLUS
                ANSWERS '7-10' FROM FILE MEDLINE
                ANSWERS '11-15' FROM FILE BIOSIS
                ANSWER '16' FROM FILE EMBASE
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Inventor search results

=> d L91 1-16 ibib ab

L91 ANSWER 1 OF 16 HCAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2003:733668 HCAPLUS Full-text
TITLE: NH_4^+ conductance in Xenopus laevis oocytes. III. Effect of NH_3
AUTHOR(S): Boldt, Matthias; Burckhardt, Gerhard; Burckhardt, Birgitta Christina
CORPORATE SOURCE: Zentrum Physiologie und Pathophysiologie, Abteilung Vegetative Physiologie und Pathophysiologie, Georg-August-Universitaet Goettingen, Goettingen, 37073, Germany
SOURCE: Pfluegers Archiv (2003), 446(6), 652-657
CODEN: PFLABK; ISSN: 0031-6768
PUBLISHER: Springer-Verlag
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Exposure of Xenopus laevis oocytes to NH_4Cl caused intracellular acidification, cell membrane depolarization and the generation of an inward current. To determine the contribution of uncharged NH_3 and pos. charged NH_4^+, the NH_4Cl-induced inward current was measured in the presence of increasing [NH_3] at constant [NH_4Cl] (10 mM) or increasing [NH_4Cl] at constant [NH_3] (0.045 mM) with pH varying in both cases. At -70 mV, the NH_4Cl-induced current was barely detectable at pH 6.5, 0.01 mM NH_3, but increased successively at pH 7.5, 0.1 mM NH_3 and pH 8.5, 1 mM NH_3. In contrast, NH_4Cl-associated currents were independent of changes of the [NH_4Cl] at constant [NH_3] and variable pH. Similar results with respect to acidification, depolarization and inward current in response to concentration and pH changes were obtained with trimethylamine HCl. Increasing concns. of the weak acid propionate led to a reduction of the NH_4Cl-induced current. These data suggest that NH_3 entry may induce local alkalinization that, in turn, may trigger the opening of a conductance for NH_4^+ or trimethylamine-H^+ entry.
REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L91 ANSWER 2 OF 16 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2007:462029 HCAPLUS Full-text
DOCUMENT NUMBER: 146:448293
TITLE: Creatine salts and method of making same
INVENTOR(S): Boldt, Matthias
PATENT ASSIGNEE(S): Starmark Laboratories, USA
SOURCE: U.S. Pat. Appl. Publ., 4 pp., Cont.-in-part of U.S. Ser. No. 740,263.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2007093677	A1	20070426	US 2006-521699	20060915
US 7301051	B2	20071127		
US 2004133040	A1	20040708	US 2003-740263	20031218
US 7109373	B2	20060919		
PRIORITY APPLN. INFO.:			US 2002-434245P P	20021218
			US 2003-740263 A2	20031218

AB Disclosed are creatine salts with an anion of dicarboxylic acid, such as ketoglutaric acid and succinic acid. The creatine salts are stable and may provide improved bioavailability.

L91 ANSWER 3 OF 16 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:99181 HCAPLUS Full-text
DOCUMENT NUMBER: 142:183472
TITLE: Nutrient compositions and methods for sustenance and promotion of positive metabolic energy levels in a targeted manner
INVENTOR(S): Boldt, Matthias
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 7 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005027005	A1	20050203	US 2003-633232	20030802
PRIORITY APPLN. INFO.:			US 2003-633232	20030802

AB Nutrient compns. and methods that sustain and promote pos. metabolic energy levels in a targeted manner are disclosed. Methods utilize endogenous energy stores (fat oxidation), increase use of those stores (increasing transport rate), increase available energy (increasing the ability to perform ADP to ATP phosphorylation,) as well as decrease catabolism and increase protein synthesis. Compns. are also disclosed, and include mono- or dicreatine-β-hydroxy β-methylbutyrate (HMB) salt; putrescine dihydrochloride; alanine; L-glutamine, which may be combined with alanine in a 1:2 to 2:1 mol. ratio; trimethylglycine; and guanidinopropionic acid.

L91 ANSWER 4 OF 16 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:98865 HCAPLUS Full-text
DOCUMENT NUMBER: 142:162689
TITLE: Weight control compositions and methods for fat loss and lean body mass maintenance
INVENTOR(S): Boldt, Matthias
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 6 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005025844	A1	20050203	US 2003-633233	20030802
PRIORITY APPLN. INFO.:			US 2003-633233	20030802

AB The present invention provides compns. and methods that assist in providing weight control. Compns. comprise caffeine, an adrenergic amine (e.g. synephrine, hordenine, octopamine, tyramine and N-methyltyramine,) forskolin, Guggulsterones, an α-2 receptor antagonist (e.g. yohimbine) and a vinca alkaloid (e.g. vinpocetine). Black pepper extract may be added as well in

various alternative embodiments. Methods utilizing administration of nutrient compns. are disclosed as well.

L91 ANSWER 5 OF 16 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:550807 HCAPLUS Full-text
DOCUMENT NUMBER: 141:88865
TITLE: Preparation of creatine salts of dicarboxylic acids
INVENTOR(S): Boldt, Matthias
PATENT ASSIGNEE(S): San Corporation, USA
SOURCE: U.S. Pat. Appl. Publ., 4 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004133040	A1	20040708	US 2003-740263	20031218
US 7109373	B2	20060919		
US 2007093677	A1	20070426	US 2006-521699	20060915
US 7301051	B2	20071127		
PRIORITY APPLN. INFO.:			US 2002-434245P	P 20021218
			US 2003-740263	A2 20031218

OTHER SOURCE(S): MARPAT 141:88865
AB Creatine salts of dicarboxylic acids [H2NC:NHN(CH3)CH2CO2H]2 A (A = an anion of a dicarboxylic acid; e.g., dicreatine maleate) are prepared by the neutralization of the dicarboxylic acid with an alc. solution of creatine or its monohydrate.
REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L91 ANSWER 6 OF 16 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1980:440841 HCAPLUS Full-text
DOCUMENT NUMBER: 93:40841
TITLE: A sensitive dual wavelength microspectrophotometer for the measurement of tissue fluorescence and reflectance
AUTHOR(S): Boldt, M.; Harbig, K.; Weidemann, G.; Luebbers, D. W.
CORPORATE SOURCE: Max-Planck-Inst. Systemphysiol., Dortmund, D-4600, Fed. Rep. Ger.
SOURCE: Pfluegers Archiv (1980), 385(2), 167-73
CODEN: PFLABK; ISSN: 0031-6768
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The construction of a microscope photometer using prefabricated elements is described. To illuminate the tissue, a Leitz Ultropac is applied. To enlarge the wavelength range, its illuminating glass lens is replaced by an Acryl glass zonal lens. Two sep. light channels with sep. lamps, monochromators and photomultipliers allow the measurement of fluorescence excitation and emission spectra as well as of reflection spectra. By chopping the light, light pulses and dark current are measured 8.33 times a sec. By an integration circuit the signal-to-noise ratio for small signals is improved. The instrument detects the increase of 4 ng/mL NADH (pH 7.39) in an area 0.2 mm2.

L91 ANSWER 7 OF 16 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 1998080667 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 9419397

TITLE: Calcium and colorectal epithelial cell proliferation in ulcerative colitis.
AUTHOR: Bostick R M; Boldt M; Darif M; Wood J R; Overn P; Potter J D
CORPORATE SOURCE: Department of Public Health Sciences-Epidemiology, Bowman Gray School of Medicine, Wake Forest University, Winston-Salem, North Carolina 27157, USA.
SOURCE: Cancer epidemiology, biomarkers & prevention : a publication of the American Association for Cancer Research, cosponsored by the American Society of Preventive Oncology, (1997 Dec) Vol. 6, No. 12, pp. 1021-7.
Journal code: 9200608. ISSN: 1055-9965.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199801
ENTRY DATE: Entered STN: 30 Jan 1998
Last Updated on STN: 30 Jan 1998
Entered Medline: 22 Jan 1998

AB In persons at higher risk for colon cancer (e.g., those with sporadic adenoma or ulcerative colitis), compared to those at lower risk, colonic epithelial cell proliferation kinetics are altered. We have shown previously that calcium supplementation appears to normalize the distribution of proliferating cells without affecting the proliferation rate in the colorectal mucosa of sporadic adenoma patients. In a pilot randomized, double-blind, placebo-controlled, clinical trial conducted concurrently with our previously published sporadic adenoma trial, we tested whether calcium supplementation can also modulate cell proliferation kinetics in patients with ulcerative colitis. Ulcerative colitis patients (n = 31) were randomized to placebo or 2.0 g of supplemental calcium daily. Colorectal epithelial cell proliferation was determined by immunohistochemical detection of proliferating cell nuclear antigen labeling of cells in "nonprep" rectal biopsies taken at randomization and after 2 months treatment. All biopsies were scored by one reviewer. Differences in mean follow-up minus baseline labeling index (LI; the proportion of colon crypt epithelial cells that were labeled) and in the phi(h) (proportion of labeled cells that were in the upper 40% of the crypts) were compared with analysis of covariance. Pill-taking adherence was 97%. Biopsy-scoring reliability was high (r = 0.89). The pooled baseline LI and phi(h) were 6.3% and 5.6%, respectively. The LI in the calcium group decreased by 0.5% (proportionately, 3%) more than in the placebo group (P = 0.91). Similarly, the phi(h) in the calcium group decreased by 0.3% (proportionately, 10%) more than in the placebo group (P = 0.85). This pilot study does not suggest that 2.0 g of calcium as calcium carbonate daily can substantially normalize either the rate or distribution of proliferating cells over a 2-month period in the colon crypts of patients with ulcerative colitis; a more definitive answer to the question of whether calcium may be effective would require a study with a larger sample size and/or other study design modifications.

L91 ANSWER 8 OF 16 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 89333462 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 2502902
TITLE: Significance of nitroglycerin-induced hypotension with inferior wall acute myocardial infarction.
AUTHOR: Ferguson J J; Diver D J; Boldt M; Pasternak R C
CORPORATE SOURCE: Charles A. Dana Research Institute, Boston, Massachusetts.

CONTRACT NUMBER: HL-07374 (NHLBI)
SOURCE: The American journal of cardiology, (1989 Aug 1)
Vol. 64, No. 5, pp. 311-4.
Journal code: 0207277. ISSN: 0002-9149.
PUB. COUNTRY: United States
DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198908
ENTRY DATE: Entered STN: 9 Mar 1990
Last Updated on STN: 3 Feb 1997
Entered Medline: 28 Aug 1989

AB Up to 60% of patients with inferior wall acute myocardial infarction (AMI) develop hypotension. In many cases, profound hypotension is precipitated by the administration of nitroglycerin. To test the hypothesis that this hypotensive response to nitroglycerin may be related to right ventricular (RV) involvement, we compared 20 patients with electrocardiographic and enzyme-documented inferior wall AMI and marked hypotension (greater than 30 mm Hg decrease in systolic blood pressure, with symptoms) after nitrate administration, to 20 patients with documented inferior AMI, but without hypotension after administration of nitroglycerin. The presence of RV involvement was determined by electrocardiographic criteria of 1 mm of ST-segment elevation in at least 2 right precordial chest leads. Fifteen of the 20 patients who demonstrated a marked hypotensive response to nitroglycerin had evidence of RV involvement, while in 18 of the 20 patients without hypotension after nitrates there was no evidence of RV involvement. In a separate analysis of 28 patients with documented RV involvement in an inferior AMI, 20 developed hypotension in response to nitrates. Thus, in the setting of an inferior AMI, a marked hypotensive response to nitrates suggests the presence of RV involvement. Moreover, hypotension after nitrate administration may be anticipated in patients with known RV infarction, and in such patients, nitrates should be administered carefully.

L91 ANSWER 9 OF 16 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 88087885 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 3121673
TITLE: Experimental chemotherapy-induced skin necrosis in swine. Mechanistic studies of anthracycline antibiotic toxicity and protection with a radical dimer compound.
AUTHOR: Averbuch S D; Boldt M; Gaudiano G; Stern J B; Koch T H; Bachur N R
CORPORATE SOURCE: Division of Cancer Treatment, National Cancer Institute, Bethesda, Maryland 20892.
CONTRACT NUMBER: CA-24665 (NCI)
SOURCE: The Journal of clinical investigation, (1988 Jan)
Vol. 81, No. 1, pp. 142-8.
Journal code: 7802877. ISSN: 0021-9738.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198802
ENTRY DATE: Entered STN: 5 Mar 1990
Last Updated on STN: 3 Mar 2000
Entered Medline: 9 Feb 1988

AB The reactivity of antitumor anthracycline and mitomycin C antibiotics with the oxomorpholinyl radical dimers, bi(3,5,5-trimethyl-2-oxomorpholin-3-yl) (TM3) and bi(3,5-dimethyl-5-hydroxymethyl-2-oxomorpholin-3-yl) (DHM3), was studied in vitro. The oxomorpholinyl radical reduced daunorubicin to a quinone methide intermediate that reacted with solvent to form 7-deoxydaunorubicinone. The solvolysis reaction followed first order kinetics, and the reactivity rate constants (k2) measured for seven anthracycline analogues ranged from 2 X 10(-2) s-1 to 8.0 X 10(-4) s-1. The chemical reactivity of each anthracycline quinone methide correlated with the total skin toxicity caused by the respective parent anthracycline following injection into swine skin. Microscopic examination of experimental lesions in swine skin resemble those observed in humans after inadvertant chemotherapy extravasation. Hydrocortisone sodium succinate was not effective for the treatment of doxorubicin-induced skin necrosis, whereas DHM3 was effective for the treatment of skin necrosis caused by all seven anthracyclines and by the quinone containing antibiotic, mitomycin C.

L91 ANSWER 10 OF 16 MEDLINE on STN
ACCESSION NUMBER: 86079900 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 10274736
TITLE: Towards the development of a systematic approach to suicide prevention: the Alberta model.
AUTHOR: Boldt M
SOURCE: Canada's mental health, (1985 Jun) Vol. 33, No. 2, pp. 2-4.
Journal code: 0070157. ISSN: 0008-2791.
PUB. COUNTRY: Canada
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Health
ENTRY MONTH: 198602
ENTRY DATE: Entered STN: 23 Feb 2001
Last Updated on STN: 23 Feb 2001
Entered Medline: 12 Feb 1986

AB The author outlines a model recently adopted by the Province of Alberta to provide suicide prevention, intervention and postvention services. Based on the proposals of a Provincial Task Force, the model features interrelated programs of outreach, education and training, research, and fund-raising. It is designed to make use of community resources in an efficient and coordinated manner, attacking the problem on several fronts.

L91 ANSWER 11 OF 16 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 3
ACCESSION NUMBER: 1989:231889 BIOSIS Full-text
DOCUMENT NUMBER: PREV198936110373; BR36:110373
TITLE: LEUCODAUNOMYCIN A TAUTOMER OF DAUNOMYCIN HYDROQUINONE.
AUTHOR(S): BIRD D M [Reprint author]; BOLDT M; KOCH T H
CORPORATE SOURCE: DEP CHEM BIOCHEM, UNIV COLO, BOULDER, CO 80309-0215, USA
SOURCE: Journal of the American Chemical Society, (1989) Vol. 111, No. 3, pp. 1148-1150.
CODEN: JACSAT. ISSN: 0002-7863.
DOCUMENT TYPE: Article
FILE SEGMENT: BR
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 11 May 1989
Last Updated on STN: 11 May 1989

L91 ANSWER 12 OF 16 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on

STN
ACCESSION NUMBER: 1989:270692 BIOSIS Full-text
DOCUMENT NUMBER: PREV198988006774; BA88:6774
TITLE: FORMATION AND REACTION OF THE QUINONE METHIDE FROM REDUCTIVE CLEAVAGE OF THE ANTITUMOR DRUG MENOGARIL.
AUTHOR(S): BOLDT M [Reprint author]; GAUDIANO G; HADDADIN M J; KOCH T H
CORPORATE SOURCE: DEP CHEM BIOCHEM, UNIV COLORADO, BOULDER, COLO 80309-0215, USA
SOURCE: Journal of the American Chemical Society, (1989) Vol. 111, No. 6, pp. 2283-2292. CODEN: JACSAT. ISSN: 0002-7863.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 6 Jun 1989
Last Updated on STN: 6 Jun 1989

AB Anaerboic reduction of menogaril (1), a semisynthetic antitumor drug in clinical trials, with d,l-bi(3,5,5-trimethyl-2-oxomorpholin-3-yl) (TM-3 dimer) in methanol gave 7-deoxynogarol (5) and stereoisomers of bi(7-deoxynogarol-7-yl) (6) and,, in the presence of N-acetylcysteine, 7-(N-acetylcysteinyl)-7-deoxynogarol (10) via an observed quinone methide intermediate (8). In the presence of excess reducing agent, 5 was formed relatively rapidly as the major product in its hydroquinone state. The rate-controlling step, tautomerization of the quinone methide, was autocatalyzed; the product, the hydroquinone of 5, catalyzed the reaction. In fact, several anthracycline-derived hydroquinone were effective catalysts. Uncatalyzed tautomerization of the quinone methide yielded little if any 5, in contrast with facile unimolecular formation of 7-deoxyaglycons from reduction of other anthracyclines. In the absence or presence of excess reducing agent, the rate of formation of 6 or formation of 6 in its bishydroquinone state, respectively, was second order in quinone methide concentration and relatively slow. The rate constants for the autocatalyzed tautomerization and the dimerization of the quinone methide are 27 ± 2 and 11 ± 1 M-1 s-1, respectively. Reduction of menogaril in aqueous medium gave predominantly 7-deoxynogarol (5) relatively rapidly with excess reducing agent and a mixture of 5 and the aglycon dimer 6 slowly with substoichiometric amounts of reducing agent. Under both sets of conditions, the quinone methide transient was not observed. Reduction in aqueous medium with 0.3 equiv of reducing agent in the presence of N-acetylcysteine gave high yields of adduct 10, suggesting a relatively long lifetime for the unobservable quinone methide transient even in aqueous medium in the absence of hydroquinones and reactive nucleophiles. A possible in vivo consequence of the relatively slow uncatalyzed tautomerization of the quinone methide is efficient nucleophilic trapping.

L91 ANSWER 13 OF 16 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 1988:319078 BIOSIS Full-text
DOCUMENT NUMBER: PREV198835024412; BR35:24412
TITLE: FORMATION AND AUTOCATALYTIC DESTRUCTION OF THE QUINONE METHIDE FROM REDUCTIVE CLEAVAGE OF MENOGARIL.
AUTHOR(S): BOLDT M [Reprint author]; GAUDIANO G; HADDADIN M J; KOCH T H
CORPORATE SOURCE: DEP CHEM BIOCHEM, UNIV COLO, BOULDER, COLO 80309-0215, USA
SOURCE: Journal of the American Chemical Society, (1988) Vol. 110, No. 10, pp. 3330-3332. CODEN: JACSAT. ISSN: 0002-7863.
DOCUMENT TYPE: Article
FILE SEGMENT: BR

LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 11 Jul 1988
Last Updated on STN: 11 Jul 1988

L91 ANSWER 14 OF 16 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 1987:339444 BIOSIS Full-text
DOCUMENT NUMBER: PREV198784048387; BA84:48387
TITLE: SUBSTITUENT EFFECTS ON THE REDOX CHEMISTRY OF ANTHRACYCLINE ANTITUMOR DRUGS.
AUTHOR(S): BOLDT M [Reprint author]; GAUDIANO G; KOCH T H
CORPORATE SOURCE: DEP CHEM BIOCHEM, UNIV COLO, BOULDER, COLO 80309-0215, USA
SOURCE: Journal of Organic Chemistry, (1987) Vol. 52, No. 11, pp. 2146-2153.
CODEN: JOCEAH. ISSN: 0022-3263.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 8 Aug 1987
Last Updated on STN: 8 Aug 1987
AB Reduction of 11-deoxydaunomycin (8), adriamycin (1), 4-demethoxydaunomycin (9), and 4-methoxy-6-deoxydaunomycin (10) with meso- and d,l-3,3',5,5,5',5'-hexamethyl-2,2'-dioxo-3,3'-bimorpholinyl (3 and 4) is described. Quinone methide intermediates from glycosidic cleavage of reduced 1, 8, and 9 were characterized by UV-vis spectroscopy and the rate constants for their tautomerization to the respective 7-deoxyaglycons were determined. These rate constants together with those from earlier measurements, ranging from 0.013 to 0.000095 s-1, establish an order of nucleophilicity of the quinone methides from reductive glycosidic cleavage of five anthracyclines of biological interest. The dimerization of the quinone methide from reduction of 11-deoxydaunomycin was established and the rate constant determined for comparison with the rate constant for dimerization of the quinone methide from reduction of aclacinomycin A. Reduction of 10 did not yield glycosidic cleavage but only catalysis of the disproportionation of 4 most likely by hydride transfer from the hydroquinone of 10 to 5,6-dihydro-3,5-5-trimethyl-1,4-oxazin-2-one (5), the product of oxidation of 4. The rate constant for hydride transfer was measured as a function of pH and compared with the rate constant for hydride transfer from 7-deoxydaunomycinone hydroquinone to 5.

L91 ANSWER 15 OF 16 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 1986:30072 BIOSIS Full-text
DOCUMENT NUMBER: PREV198630030072; BR30:30072
TITLE: NITROGLYCERIN INDUCED HYPOTENSION WITH ACUTE INFERIOR MYOCARDIAL INFARCTION A MARKER OF RIGHT VENTRICULAR INVOLVEMENT?.
AUTHOR(S): FERGUSON J J [Reprint author]; DIVER D J; BOLDT M ; PASTERNAK R C
CORPORATE SOURCE: HARVARD-THORNDIKE LAB, BETH ISRAEL HOSP, BOSTON, MASS, USA
SOURCE: American Heart Association Monograph, (1985) No. 114, pp. III-460.
Meeting Info.: JOINT PROCEEDINGS OF THE 58TH SCIENTIFIC SESSIONS OF THE AMERICAN HEART ASSOCIATION, THE SCIENTIFIC SESSIONS FOR NURSES, AND THE 39TH ANNUAL MEETING OF THE COUNCIL ON ARTERIOSCLEROSIS OF THE AMERICAN SOCIETY FOR THE STUDY OF ARTERIOSCLEROSIS, WASHINGTON, D.C., USA, NOV. 11-14, 1985. AM HEART ASSOC MONOGR.
CODEN: AHMOAH. ISSN: 0065-8499.
DOCUMENT TYPE: Conference; (Meeting)

FILE SEGMENT: BR
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 25 Apr 1986
Last Updated on STN: 25 Apr 1986

L91 ANSWER 16 OF 16 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2002359508 EMBASE Full-text
TITLE: Characterizing and developing strategies for the treatment of community-acquired pneumonia at a community hospital.
AUTHOR: Fok M.C.; Kanji Z.; Mainra R.; Boldt M.
CORPORATE SOURCE: M.C. Fok, Vancouver Hospital/Health Sci. Ctr., University of BC Hospital Site, Pharmacy Department, 2211 Wesbrook Mall, Vancouver, BC V6T 2B5, Canada. mfok@vanhosp.bc.ca
SOURCE: Canadian Respiratory Journal, (Jul 2002) Vol. 9, No. 4, pp. 247-252.
Refs: 11
ISSN: 1198-2241 CODEN: CRJOFV
COUNTRY: Canada
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
036 Health Policy, Economics and Management
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 24 Oct 2002
Last Updated on STN: 24 Oct 2002

AB Background: Patients admitted to Lions Gate Hospital, North Vancouver, British Columbia, with a primary diagnosis of community-acquired pneumonia (CAP) have a mean length of stay (LOS) of 9.1 days compared with 7.9 days for peer group hospitals. This difference of 1.2 days results in an annual potential savings of 406 bed days and warranted an investigation into the management of CAP. Objective: To characterize and provide recommendations for the management of CAP. Methods: A retrospective chart review of patients admitted with a primary diagnosis of CAP between May 1, 2000 and August 31, 2000. Results: Fifty-one patients were included in the study, with a mean LOS of 9.9 days and a median LOS of five days. Based on pneumonia severity index scores calculated for each patient, eight patients (16%) were admitted inappropriately. Initial empirical antibiotic choices were consistent with the Canadian CAP guidelines in 27 patients (53%), with inconsistencies arising mainly because cephalosporin or azithromycin monotherapy regimens were prescribed. Step-down from intravenous to oral antibiotics occurred in approximately 20 patients (39%). An additional 12 patients (24%) could have undergone step-down, and step-down was not applicable in 19 patients (37%). The potential annual cost avoidance from implementing admission criteria based on a pneumonia severity index score, applying step-down criteria and promoting early discharge criteria was estimated to be $220,000. Conclusions: Considerable variability exists in the treatment of CAP. A CAP preprinted order sheet was developed to address the issues identified in the present study and provide consistency in the management of CAP at Lions Gate Hospital.

Text search history

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=> d his L73

     (FILE 'HCAPLUS' ENTERED AT 13:59:36 ON 28 NOV 2007)
L73          14 S L71 AND L72

=> d que L73
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L3            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  56-85-9/RN
L4            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  107-43-7/RN
L5            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  333-93-7/RN
L6            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  353-09-3/RN
L7            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  835598-36-2/RN
L8            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  835598-38-4/RN
L9            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  625-08-1/RN
L10           1 SEA FILE=REGISTRY ABB=ON  PLU=ON  57-00-1/RN
L11           1 SEA FILE=REGISTRY ABB=ON  PLU=ON  110-60-1/RN
L12           1 SEA FILE=REGISTRY ABB=ON  PLU=ON  107-43-7/RN
L13       45354 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L2
L14       26751 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L3
L15        5908 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L4
L16         245 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L5
L17         369 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L6
L18           2 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L7
L19           1 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L8
L20         370 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L9
L21        6901 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L10
L22       13138 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L11
L23        5908 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L12
L24         790 SEA FILE=HCAPLUS ABB=ON  PLU=ON  ((MONO?)(3A)CREATIN?)
L25          15 SEA FILE=HCAPLUS ABB=ON  PLU=ON  DICREATIN?
L27         125 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (GUANIDIN?(3A)PROPION?)
L28         728 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (HYDROXY?)(3A)(METHYLBUTYR?)
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                L17 OR L18 OR L19)) AND (ENTER? OR PARENTER?)
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                L25
L31       15982 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L16 OR L22 OR PUTRESCINE?
L32          20 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (PUTRESCIN?(2A)HYDROCHLOR?)
L33      146242 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L2 OR ALANINE?
L34       52786 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L3 OR GLUTAMINE
L35        6019 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L15 OR L23 OR TRIMETHYLGLYCINE
                 OR (TRIMETHYL(2A)GLYCINE)
L36         616 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L17 OR L27 OR GUANIDINOPROPION
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                AND L36
L39           8 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L30 AND L31 AND L33 AND L34
L40           2 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L39 AND L35
L41           1 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L39 AND L36
L42           0 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L32 AND L37
L43           2 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L18 OR L19
L44           2 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L43 AND (ADMINIST? OR ENTER?
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L50           1 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L13 AND L14 AND L15 AND L16
                AND L17
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                L42 OR L43 OR L44)) OR L47 OR L50 OR L51
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L59          15 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L55 OR L58
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L63          45 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L37 AND L30
L64          36 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L63 AND (ADMINIST? OR THERAP?
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                OR TREAT? OR PERFORM? OR SPORT? OR DIET? OR ENTER? OR PARENTER?
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L68           2 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L67 AND L30
L69          18 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L59 OR L61 OR L65 OR L68
L70           1 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L64 AND L66
L71          18 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L69 OR L70
L72             QUE  ABB=ON  PLU=ON  AY<2004 OR PY<2004 OR PRY<2004 OR RE
                VIEW/DT
L73          14 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L71 AND L72


=> d his L85

     (FILE 'MEDLINE, BIOSIS, EMBASE, DRUGU' ENTERED AT 14:52:16 ON 28 NOV 2007)
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=> d que L85
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L3            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  56-85-9/RN
L4            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  107-43-7/RN
L5            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  333-93-7/RN
L6            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  353-09-3/RN
L7            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  835598-36-2/RN
L8            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  835598-38-4/RN
L9            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  625-08-1/RN
L10           1 SEA FILE=REGISTRY ABB=ON  PLU=ON  57-00-1/RN
L11           1 SEA FILE=REGISTRY ABB=ON  PLU=ON  110-60-1/RN
L12           1 SEA FILE=REGISTRY ABB=ON  PLU=ON  107-43-7/RN
L13       45354 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L2
L14       26751 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L3
L15        5908 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L4
L16         245 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L5
L17         369 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L6
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L19           1 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L8
L20         370 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L9
L21        6901 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L10
L22       13138 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L11
L23        5908 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L12
L24         790 SEA FILE=HCAPLUS ABB=ON  PLU=ON  ((MONO?)(3A)CREATIN?)
L25          15 SEA FILE=HCAPLUS ABB=ON  PLU=ON  DICREATIN?
L27         125 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (GUANIDIN?(3A)PROPION?)
L28         728 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (HYDROXY?)(3A)(METHYLBUTYR?)
L29        2097 SEA FILE=HCAPLUS ABB=ON  PLU=ON  ((L13 OR L14 OR L15 OR L16 OR
                L17 OR L18 OR L19)) AND (ENTER? OR PARENTER?)
L30       30974 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L21 OR CREATINE? OR L24 OR
                L25
L31       15982 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L16 OR L22 OR PUTRESCINE?
L32          20 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (PUTRESCIN?(2A)HYDROCHLOR?)
L33      146242 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L2 OR ALANINE?
L34       52786 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L3 OR GLUTAMINE
L35        6019 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L15 OR L23 OR TRIMETHYLGLYCINE
                 OR (TRIMETHYL(2A)GLYCINE)
L36         616 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L17 OR L27 OR GUANIDINOPROPION
                ?
L37          45 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L30 AND (L20 OR L28)
L38           0 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L32 AND L33 AND L34 AND L35
                AND L36
L39           8 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L30 AND L31 AND L33 AND L34
L40           2 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L39 AND L35
L41           1 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L39 AND L36
L42           0 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L32 AND L37
L43           2 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L18 OR L19
L44           2 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L43 AND (ADMINIST? OR ENTER?
                OR PARENTER? OR SUPPLEM? OR ADDITI? OR PERFORMAN? OR SPORT?)
L47           0 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L20 AND L21 AND L22 AND L23
L50           1 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L13 AND L14 AND L15 AND L16
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L51           0 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L30 AND L32
L52           9 SEA FILE=HCAPLUS ABB=ON  PLU=ON  ((L38 OR L39 OR L40 OR L41 OR
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L53           0 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L29 AND L32
L54           5 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L32 AND (ADMINIST? OR THERAP?
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L55          14 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (L52 OR L53 OR L54)
L56        1915 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L35 AND (ADMINIST? OR THERAP?
                OR TREAT? OR PERFORM? OR SPORT? OR DIET? OR ENTER? OR PARENTER?
                )
L57         260 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L36 AND (ADMINIST? OR THERAP?
                OR TREAT? OR PERFORM? OR SPORT? OR DIET? OR ENTER? OR PARENTER?
                )
L58           2 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L56 AND L57
L59          15 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L55 OR L58
L60          36 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L37 AND (ADMINIST? OR THERAP?
                OR TREAT? OR PERFORM? OR SPORT? OR DIET? OR ENTER? OR PARENTER?
                )
L61           3 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L60 AND L33 AND L34
L63          45 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L37 AND L30
L64          36 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L63 AND (ADMINIST? OR THERAP?
                OR TREAT? OR PERFORM? OR SPORT? OR DIET? OR ENTER? OR PARENTER?
                )
L65           1 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L64 AND L31
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L66        5865 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L31 AND (ADMINIST? OR THERAP?
                OR TREAT? OR PERFORM? OR SPORT? OR DIET? OR ENTER? OR PARENTER?
                )
L67          70 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L66 AND L16
L68           2 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L67 AND L30
L69          18 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L59 OR L61 OR L65 OR L68
L70           1 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L64 AND L66
L71          18 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L69 OR L70
L72             QUE  ABB=ON  PLU=ON  AY<2004 OR PY<2004 OR PRY<2004 OR RE
                VIEW/DT
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                DICREATIN?) AND ALANIN? AND GLUTAM?
L77           9 SEA (PUTRESCIN?) AND (CREATIN? OR MONO(3N) CREATIN? OR
                DICREATIN?) AND GUANIDIN?
L78          27 SEA (L75 OR L76 OR L77)
L79          48 SEA (PUTRESCIN?) AND (CREATIN? OR MONO(3N) CREATIN? OR
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                OR TREAT?)
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L81           6 SEA L80 AND ALANIN? AND GLUTAM?
L82          17 SEA L80 AND AMINO?
L83          20 SEA L81 OR L82
L84          32 SEA L78 OR L83
L85          30 SEA L84 AND L72


=> dup rem L73 L85
PROCESSING COMPLETED FOR L73
PROCESSING COMPLETED FOR L85
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                ANSWERS '15-19' FROM FILE MEDLINE
                ANSWERS '20-26' FROM FILE BIOSIS
                ANSWERS '27-34' FROM FILE EMBASE
                ANSWERS '35-36' FROM FILE DRUGU
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Text search results

=> d L92 1-14 ibib ed abs hitind

L92 ANSWER 1 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 2
ACCESSION NUMBER: 1997:521359 HCAPLUS Full-text
DOCUMENT NUMBER: 127:171325
TITLE: Glutamate uptake is inhibited by L-arginine in mitochondria isolated from rat cerebrum
AUTHOR(S): Dolinska, Monika; Albrecht, Jan
CORPORATE SOURCE: Department of Neurotoxicology, Medical Research Centre, Polish Academy of Sciences, Warsaw, 02-106, Pol.
SOURCE: NeuroReport (1997), 8(9-10), 2365-2368
CODEN: NERPEZ; ISSN: 0959-4965
PUBLISHER: Rapid Science Publishers
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 15 Aug 1997
AB Uptake of L-[14C]glutamate (L-[14C]GLU) into nonsynaptic mitochondria isolated from rat cerebral hemispheres was measured in the presence of potential modulators of amino acid transport. The L-GLU carrier agonist 0.2 mM L-aspartate (L-ASP) virtually abolished L-GLU uptake (ASP/GLU concentration ratio, 1:1). L-Arginine (L-ARG) inhibited L-GLU uptake in a dose dependent manner over the concentration range 0.1-5 mM to maximum inhibition of 85%. Putrescine or ammonia had no effect, whereas 5 mM creatine and the NO generator, 5 mM sodium nitroprusside, increased the uptake by 73% and 57%, resp. D-ARG was three times less effective in inhibiting L-GLU uptake than L-ARG at 5 mM concentration The L-amino acids ornithine, lysine, histidine, tyrosine, phenylalanine, proline, leucine, isoleucine, tryptophan, glycine, methionine, valine, serine, taurine, alanine or cysteine did not affect the uptake when added in concns. of 2-5 mM. A 14% inhibition of L-GLU uptake was noted in the presence of L- glutamine (L-GLN) (2 mM) or a dicarboxylate carrier ligand, α-ketoglutarate (α-KG) (5 mM), and a 30% inhibition with a dicarboxylate carrier inhibitor phenylsuccinate (PhSc) (5 mM). The results suggest that L-ARG functions as a specific endogenous modulator of cerebral mitochondrial L-GLU transport.
CC 1-8 (Pharmacology)
Section cross-reference(s): 13
IT 52-90-4, L-Cysteine, biological studies 56-40-6, Glycine, biological studies 56-41-7, L-Alanine, biological studies
56-45-1, L-Serine, biological studies 56-84-8, L-Aspartic acid, biological studies 56-87-1, L-Lysine, biological studies 60-18-4, L-Tyrosine, biological studies 61-90-5, L-Leucine, biological studies 63-68-3, L-Methionine, biological studies 63-91-2, L-Phenylalanine, biological studies 70-26-8, L-Ornithine 71-00-1, L-Histidine, biological studies 72-18-4, L-Valine, biological studies 73-22-3, L-Tryptophan, biological studies 73-32-5, L-Isoleucine, biological studies 107-35-7, Taurine 110-60-1, Putrescine
147-85-3, L-Proline, biological studies 157-06-2, D-Arginine 328-50-7
635-51-8 7664-41-7, Ammonia, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(effect of amino acids, their metabolites, and derivs. on L-glutamate uptake in rat cerebral nonsynaptic mitochondria)
IT 57-00-1, Creatine 10102-43-9, Nitrogen oxide (NO), biological studies
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(glutamate uptake is inhibited by L-arginine in mitochondria isolated from rat cerebrum in relation to creatine and nitric oxide)

L92 ANSWER 2 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:1201156 HCAPLUS Full-text
DOCUMENT NUMBER: 143:446260
TITLE: Cosmetic or dermatological preparation comprising a nutrient medium phase and uses for physiological wound healing or scar reduction
INVENTOR(S): Monks, Monika; Ibanez, Sybille; Evangelisti, Carmen; Gohla, Sven
PATENT ASSIGNEE(S): Beiersdorf A.-G., Germany
SOURCE: U.S. Pat. Appl. Publ., 13 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005249691	A1	20051110	US 2004-967232	20041019 <--
US 2005287182	A1	20051229	US 2005-68052	20050301 <--
EP 1609462	A1	20051228	EP 2005-101620	20050303
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, BA, HR, IS, YU				

PRIORITY APPLN. INFO.:
DE 2003-10323510 A 20030524 <--
DE 2003-10355110 A 20031124 <--
DE 2004-102004020035A 20040422
WO 2004-EP5533 A1 20040522
US 2004-967232 A2 20041019

ED Entered STN: 11 Nov 2005
AB The invention comprises a cosmetic or dermatol. preparation comprising at least one nutrient medium phase for skin cells or corneal cells in combination with an aerogel or hydrogel matrix, containing collagens, chitosans having a degree of acetylation of at least 50% and chondroitin sulfates. The invention further comprises cell culture media as aqueous phase in combination with the gelling matrix described above in synergistic use with polyurethanes which are used for physiol. wound healing or scar reduction
IC ICM A61K007-06
ICS A61K007-11
INCL 424070130; 424070140
CC 62-4 (Essential Oils and Cosmetics)
IT 50-89-5, Thymidine, analysis 50-99-7, Glucose, analysis 52-89-1, L-Cysteine hydrochLoride 56-41-7, L-Alanine, analysis 56-84-8, L-Aspartic acid, analysis 56-85-9, L-Glutamine, analysis 56-86-0, L-Glutamic acid, analysis 56-89-3, L-Cystine, analysis 58-56-0, Pyridoxine hydrochloride 59-30-3, Folic acid, analysis 60-33-3, Linoleic acid, analysis 65-22-5, PyridoxaL hydrochLoride 67-03-8, Thiamine hydrochloride 67-48-1, Choline chloride 68-19-9, Vitamin B12 68-94-0, Hypoxanthine 70-47-3, L-Asparagine, analysis 72-18-4, L-VaLine, analysis 73-32-5, L-Isoleucine, analysis 83-88-5, Riboflavin, analysis 98-92-0, Nicotinamide 110-60-1, Putrescine 113-24-6, Sodium pyruvate 127-09-3, Sodium acetate 144-55-8, Carbonic acid monosodium salt, analysis 333-93-7 1119-34-2, L-Arginine hydrochloride 1200-22-2, Lipoic acid 7447-40-7, Potassium chloride (KCl), analysis 7558-79-4 7646-79-9, Cobalt chloride (CoCl2), analysis 7647-14-5, Sodium chloride, analysis 7720-78-7, Ferrous sulfate 7733-02-0, Zinc

sulfate 7785-87-7 24967-93-9, Chondroitin 4-sulfate 25322-46-7, Chondroitin 6-sulfate
RL: ANT (Analyte); ANST (Analytical study)
(cosmetic or dermatol. preparation comprising nutrient medium phase and uses
for physiol. wound healing or scar reduction)
IT 56-40-6, Glycine, biological studies 56-45-1, L-Serine, biological studies 57-00-1, Creatine 60-18-4, L-Tyrosine, biological studies 61-90-5, L-Leucine, biological studies 63-68-3, L-Methionine, biological studies 63-91-2, L-Phenylalanine, biological studies 72-19-5, L-Threonine, biological studies 73-22-3, L-Tryptophan, biological studies 73-24-5, Adenine, biological studies 87-89-8, myo-Inositol 107-35-7, Taurine 137-08-6, Calcium pantothenate 139-33-3 143-74-8, Phenol red 147-85-3, L-Proline, biological studies 303-98-0, Coenzyme Q10 541-15-1, Carnitine 645-35-2, L-Histidine hydrochloride 657-27-2, L-Lysine hydrochloride 1344-09-8 7365-45-9, HEPES 7446-70-0, Aluminum chloride (AlCl3), biological studies 7558-80-7 7718-54-9, Nickel chloride (NiCl2), biological studies 7758-98-7, Copper sulfate, biological studies 7772-99-8, Tin chloride (SnCl2), biological studies 7773-01-5, Manganese chloride (MnCl2) 7783-00-8, Selenious acid 7786-30-3, Magnesium chloride (MgCl2), biological studies 7803-55-6 8012-39-3, Citrate buffer 9005-65-6, Polysorbate 80 9012-76-4, Chitosan 10043-52-4, Calcium chloride, biological studies 10141-00-1, Chromium potassium sulfate 10421-48-4 12027-67-7 15498-87-0 15596-82-4, Nickel chloride (NiCl3) 60388-02-5, Zinc orotate 130603-71-3, α Glucosyl Rutin
RL: COS (Cosmetic use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(cosmetic or dermatol. preparation comprising nutrient medium phase and uses
for physiol. wound healing or scar reduction)

L92 ANSWER 3 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:99181 HCAPLUS Full-text
DOCUMENT NUMBER: 142:183472
TITLE: Nutrient compositions and methods for sustenance and promotion of positive metabolic energy levels in a targeted manner
INVENTOR(S): Boldt, Matthias
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 7 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005027005	A1	20050203	US 2003-633232	20030802 <--

PRIORITY APPLN. INFO.: US 2003-633232 20030802 <--
ED Entered STN: 04 Feb 2005
AB Nutrient compns. and methods that sustain and promote pos. metabolic energy levels in a targeted manner are disclosed. Methods utilize endogenous energy stores (fat oxidation), increase use of those stores (increasing transport rate), increase available energy (increasing the ability to perform ADP to ATP phosphorylation,) as well as decrease catabolism and increase protein synthesis. Compns. are also disclosed, and include mono- or dicreatine-β-hydroxy β-methylbutyrate (HMB) salt; putrescine dihydrochloride; alanine; L-

glutamine , which may be combined with alanine in a 1:2 to 2:1 mol. ratio; trimethylglycine; and guanidinopropionic acid.
IC ICM A61K031-205
ICS A61K031-198
INCL 514561000; 514554000
CC 63-6 (Pharmaceuticals)
Section cross-reference(s): 17
ST nutrient creatine HMB putrescine alanine glutamine trimethylglycine guanidinopropionate
IT Exercise
(administration followed by; nutrient compns. for promotion of pos. metabolic energy levels)
IT Nutrients
(enteral; nutrient compns. for promotion of pos. metabolic energy levels)
IT Candy
Confectionery
Food additives
Gluconeogenesis
Nutrients
(nutrient compns. for promotion of pos. metabolic energy levels)
IT Nutrients
(parenteral; nutrient compns. for promotion of pos. metabolic energy levels)
IT 56-41-7, Alanine, biological studies 56-85-9, L-Glutamine, biological studies 107-43-7, Trimethylglycine 333-93-7, Putrescine dihydrochloride 353-09-3, Guanidinopropionic acid 835598-36-2 835598-38-4
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(nutrient compns. for promotion of pos. metabolic energy levels)

L92 ANSWER 4 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:964620 HCAPLUS Full-text
DOCUMENT NUMBER: 141:394813
TITLE: Dietary supplements containing extracts of cinnamon and methods of using same to enhance creatine transport
INVENTOR(S): Miller, Peter; Romero, Timothy
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 5 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004224035	A1	20041111	US 2004-823429	20040412 <--
WO 2005099455	A1	20051027	WO 2005-US12171	20050411

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,

AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1755401 A1 20070228 EP 2005-732146 20050411
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR

PRIORITY APPLN. INFO.: US 2003-462100P P 20030411 <--
US 2004-823429 A 20040412
WO 2005-US12171 W 20050411

ED Entered STN: 12 Nov 2004

AB Materials derived from cinnamon can be administered orally to humans or animals for the purpose of controlling blood glucose as well improving glucose tolerance. Controlling glucose metabolism is essential for those with impaired glucose metabolism as is the case for those with Type II diabetes where insulin function is not properly functioning. Such administration can also be used for the purpose of enhancing nutrient transport for purposes of athletic performance and controlling bodyweight and body fat levels. Similarly related, such administration can also be used for the purpose of enhancing creatine transport into excitable tissues such as skeletal muscle. The material can be administered as exts. of cinnamon and can be administered in a variety of ways including capsules, tablets, powdered beverages, bars, gels or drinks.

IC ICM A61K035-78
ICS A61K031-195

INCL 424739000; 514554000; 514565000

CC 18-6 (Animal Nutrition)
Section cross-reference(s): 63

ST dietary supplements cinnamon extn creatine

IT Dietary supplements
Human
(dietary supplements containing exts. of cinnamon and creatines and carbohydrates)

IT Carbohydrates, biological studies
RL: FFD (Food or feed use); BIOL (Biological study); USES (Uses)
(dietary supplements containing exts. of cinnamon and creatines and carbohydrates)

IT Cinnamon (horticultural common name)
(exts.; dietary supplements containing exts. of cinnamon and creatines and carbohydrates)

IT Muscle
(skeletal; dietary supplements containing exts. of cinnamon and creatines and carbohydrates for strengthening skeletal muscles)

IT 50-99-7, Dextrose, biological studies 57-00-1, Creatine 57-00-1D, Creatine, derivative 69-79-4, Maltose 94-41-7D, Chalcone, derivs. and polymers 99-20-7, Trehalose 107-43-7, Trimethyl glycine 352-97-6, Glycocyamine 353-09-3, Guanidinopropionic acid 6020-87-7, Creatine monohydrate 9050-36-6, Maltodextrin 29908-03-0 290357-35-6
RL: FFD (Food or feed use); BIOL (Biological study); USES (Uses)
(dietary supplements containing exts. of cinnamon and creatines and carbohydrates)

IT 790714-05-5, Cinnulin PF
RL: FFD (Food or feed use); NPO (Natural product occurrence); BIOL (Biological study); OCCU (Occurrence); USES (Uses)
(dietary supplements containing exts. of cinnamon and creatines and carbohydrates)

L92 ANSWER 5 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:310653 HCAPLUS Full-text

DOCUMENT NUMBER: 140:320327
TITLE: Agglomerated granular protein-rich nutritional supplement
INVENTOR(S): Lockwood, Christopher
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 16 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004071825	A1	20040415	US 2002-271239	20021015 <--
WO 2004034986	A2	20040429	WO 2003-US32646	20031015 <--
WO 2004034986	A3	20050120		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003287150	A1	20040504	AU 2003-287150	20031015 <--
PRIORITY APPLN. INFO.:			US 2002-271239 A	20021015 <--
			WO 2003-US32646 W	20031015 <--

ED Entered STN: 16 Apr 2004

AB An agglomerated granular protein-rich nutritional supplement comprises a mixture of: 13-100 percent by weight edible food proteins; 0-57 percent by weight edible carbohydrates; 0-10 percent by weight edible fats; 0-15 percent by weight edible dietary vitamins and minerals; 0-78 percent by weight edible amino acids; 0-10 percent by weight edible plant exts., and up to 4 percent by weight chondroitin sulfate, where the nutritional supplement is agglomerated and granulated in an oral unit dosage form that is directly absorbable onto the tongue or rapidly dissolvable in an aqueous liquid Specific formulations of the supplement are disclosed, for use by specific groups of individuals. A method of supplementing the nutritional intake of individuals engaged in bodybuilding and protein supplementation, meal replacement, exercise recovery or mass gaining, comprising orally administering a formulation of the protein-rich nutritional supplement. A method of augmenting the mental acuity and energy of humans, comprising orally administering another formulation of the protein-rich nutritional supplement. Methods also are disclosed for supplementing the nutritional intake of women, male bodybuilders, children and adolescents, and older adults. In all methods, the nutritional supplement is in an oral unit dosage form of either agglomerated granules or a rapidly dissolvable wafer and also includes a flavoring compound and an effervescing compound

IC ICM A23L001-30
INCL 426072000; 426656000
CC 17-6 (Food and Feed Chemistry)
Section cross-reference(s): 18, 63
IT Agglomeration
Angelica sinensis
Cranberry
Dietary fiber
Drug delivery systems

Egg white
Flavor
Flavoring materials
Food additives
Growth, animal
Health food
Human
Mucuna pruriens
Nutrients
Sweetening agents
(agglomerated granular protein-rich nutritional supplement)

IT Food
(dietetic; agglomerated granular protein-rich nutritional supplement)

IT 50-69-1, Ribose 50-81-7, Vitamin C, biological studies 50-99-7, Dextrose, biological studies 56-41-7, L-Alanine, biological studies 56-85-9, Glutamine, biological studies 56-85-9D, L-Glutamine, peptides containing 56-87-1, Lysine, biological studies 57-00-1, Creatine 57-48-7, Fructose, biological studies 58-08-2, Caffeine, biological studies 58-85-5, Biotin 59-30-3, Folic acid, biological studies 59-43-8, Thiamin, biological studies 59-67-6, Niacin, biological studies 60-18-4, Tyrosine, biological studies 61-90-5, L-Leucine, biological studies 63-91-2, Phenylalanine, biological studies 68-19-9, Vitamin B12 70-47-3, L-Asparagine, biological studies 72-18-4, Valine, biological studies 73-32-5, L-Isoleucine, biological studies 74-79-3, Arginine, biological studies 79-83-4, Pantothenic acid 83-88-5, Riboflavin, biological studies 98-79-3, Pyroglutamic acid 107-35-7, Taurine 108-01-0, DMAE 127-17-3D, Pyruvic acid, derivs. 146-48-5, Yohimbine 625-08-1, β-Hydroxy-β-methylbutyric acid 1406-16-2, Vitamin D 1406-18-4, Vitamin E 3416-24-8, Glucosamine 4151-33-1, Potassium pyruvate 4547-24-4 6020-87-7, Creatine monohydrate 6217-54-5, Docosahexaenoic acid 7235-40-7, β-Carotene 7439-89-6, Iron, biological studies 7439-95-4, Magnesium, biological studies 7439-96-5, Manganese, biological studies 7439-98-7, Molybdenum, biological studies 7440-09-7, Potassium, biological studies 7440-23-5, Sodium, biological studies 7440-47-3, Chromium, biological studies 7440-50-8, Copper, biological studies 7440-66-6, Zinc, biological studies 7440-70-2, Calcium, biological studies 7553-56-2, Iodine, biological studies 7723-14-0, Phosphorus, biological studies 7782-49-2, Selenium, biological studies 8059-24-3, Vitamin B6 9050-36-6, Maltodextrin 10284-63-6, Inzitol 10417-94-4, Eicosapentaenoic acid 11103-57-4, Vitamin A 12001-76-2, Vitamin B 12001-79-5, Vitamin K 14265-44-2, Phosphate, biological studies 16887-00-6, Chloride, biological studies 34414-83-0, Ornithine α-ketoglutarate 52009-14-0, Calcium pyruvate 55399-93-4 56038-13-2, Splenda 72087-40-2
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(agglomerated granular protein-rich nutritional supplement)

L92 ANSWER 6 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2000:749446 HCAPLUS Full-text
DOCUMENT NUMBER: 133:286430
TITLE: Pyruvic acid water-soluble and stable formulations
INVENTOR(S): Seyerl, Joachim V.
PATENT ASSIGNEE(S): SKW Trostberg A.-G., Germany
SOURCE: Brit. UK Pat. Appl., 13 pp.
CODEN: BAXXDU

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
GB 2345247	A	20000705	GB 1999-30094	19991220 <--
DE 19859771	C1	20000824	DE 1998-19859771	19981223 <--
PRIORITY APPLN. INFO.:			DE 1998-19859771 A	19981223 <--

ED Entered STN: 25 Oct 2000

AB A water-soluble, stable formulation containing pyruvic acid or its salt comprise (a) at least one saccharide or its derivative and/or one or more physiol. acceptable salts thereof, and p (b) pyruvic acid or at least one salt thereof which is different from component (a), or mixture thereof. In addition, the formulation can contain up to 20% of an alkaline earth metal carbonate and/or of an alkaline earth metal salt of an organic carboxylic acid such a as citric acid or ascorbic acid, up to 20% of other physiol. active substances such as sugar, vitamins, trace elements etc., and/or up to 20% of formulation aids. The proposed formulation is advantageous especially for the prevention and treatment of dystrophic and/or degenerative and/or inflammatory arthropathies. A pharmaceutical powder contained glucosamine 500, calcium pyruvate 750, magnesium hydrogen-L-aspartate 720, glucose 2000, and ascorbic acid 500 mg.

IC ICM A61K031-70
ICA A61K031-19; A61P003-02
CC 63-6 (Pharmaceuticals)
IT 50-21-5, Lactic acid, biological studies 50-81-7, Ascorbic acid, biological studies 56-41-7, Alanine, biological studies 56-84-8, Aspartic acid, biological studies 56-85-9, Glutamine, biological studies 57-00-1, Creatine 57-11-4D, Octadecanoic acid, salts, biological studies 70-26-8, Ornithine 74-79-3, Arginine, biological studies 77-92-9, Citric acid, biological studies 127-17-3, Pyruvic acid, biological studies 127-17-3D, Pyruvic acid, salts 131-48-6, N-Acetylneuraminic acid 328-50-7 526-95-4, Gluconic acid 541-15-1, Carnitine 625-08-1 1200-22-2, α-Lipoic acid 3416-24-8, Glucosamine 7439-89-6, Iron, biological studies 7439-98-7, Molybdenum, biological studies 7440-42-8, Boron, biological studies 7440-50-8, Copper, biological studies 7440-66-6, Zinc, biological studies 7631-86-9, Silica, biological studies 7782-49-2, Selenium, biological studies 9003-39-8, Polyvinyl pyrrolidone 9004-61-9, Hyaluronic acid 9004-67-5, Methyl cellulose 9007-27-6, Chondroitin 27750-10-3, Hydroxycitric acid 29261-87-8, Glucosamine pyruvate
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(pyruvic acid water-soluble and stable formulations)

L92 ANSWER 7 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2000:397822 HCAPLUS Full-text
DOCUMENT NUMBER: 133:140046
TITLE: Oral peptide drug delivery: polymer-inhibitor conjugates protecting insulin from enzymic degradation in vitro
AUTHOR(S): Marschutz, Michaela K.; Bernkop-Schnurch, Andreas
CORPORATE SOURCE: Institute of Pharmaceutical Technology, Center of Pharmacy, University of Vienna, Vienna, A-1090, Austria
SOURCE: Biomaterials (2000), 21(14), 1499-1507
CODEN: BIMADU; ISSN: 0142-9612
PUBLISHER: Elsevier Science Ltd.

DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 16 Jun 2000
AB A drug-carrier matrix has been developed which protects embedded insulin from degradation by the luminally secreted serine-proteases trypsin (EC 3.4.21.4), chymotrypsin (EC 3.4.21.1) and elastase (EC 3.4.21.36) in vitro. Increasing amts. of the Bowman-Birk inhibitor (BBI) and elastatinal, resp., were thereby covalently bound to the mucoadhesive polymer sodium CM-cellulose (Na-CMC). The inhibitory efficacy of resulting polymers was evaluated. On the one hand, all polymer-BBI conjugates showed a strong inhibitory activity towards trypsin and chymotrypsin whereas it was markedly lower towards elastase. The polymer-elastatinal conjugates, on the other hand, displayed a comparatively higher inhibitory activity towards elastase. In an artificial intestinal fluid containing trypsin, chymotrypsin and elastase in physiol. concns. insulin, being incorporated in unmodified Na-CMC, was rapidly degraded at 37°C. Within 1 h 98.7 ± 0.4% (mean ± SD, n = 3) of the peptide drug were thereby metabolized. On the contrary, the incorporation of insulin in a mixture of the two polymer-inhibitor conjugates CMC-BBI (40%; weight/weight) and CMC-elastatinal conjugate (60%; weight/weight) led to a peptide degradation of 22.3 ± 2.5% (mean ± SD, n = 3) within the same time period. Even after 4 h of incubation, 33.6 ± 3.2% (mean ± SD, n = 3) of the therapeutic agent remained stable towards enzymic attack. Hence, the polymer-inhibitor conjugates described in this study seem to be a useful tool in overcoming the luminal enzymic barrier in peroral insulin delivery.
CC 63-5 (Pharmaceuticals)
Section cross-reference(s): 2
IT 333-93-7DP, Putrescine hydrochloride, conjugates with CM-cellulose and elastatinal 9004-32-4DP, Sodium CM-cellulose, conjugates with Bowman-Birk inhibitor or elastatinal 37330-34-0DP, Bowman-Birk inhibitor, conjugates with CM-cellulose 51798-45-9DP, Elastatinal, conjugates with CM-cellulose and putrescine
RL: PNU (Preparation, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(polymer-inhibitor conjugates protecting insulin from enzymic degradation for oral drug delivery)
REFERENCE COUNT: 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L92 ANSWER 8 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1997:331615 HCAPLUS Full-text
DOCUMENT NUMBER: 127:46308
TITLE: Dichotomous relationship between DNA reactivity and the induction of sister chromatid exchanges in vivo and in vitro
AUTHOR(S): Labbauf, Abbas; Klopman, Gilles; Rosenkranz, Herbert S.
CORPORATE SOURCE: Department of Environmental and Occupational Health, University of Pittsburgh, Pittsburgh, PA, 15238, USA
SOURCE: Mutation Research, Fundamental and Molecular Mechanisms of Mutagenesis (1997), 377(1), 37-52
CODEN: MUREAV; ISSN: 0027-5107
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 24 May 1997
AB Structural analyses of the determinants associated with the induction of bone marrow sister chromatid exchanges in mice indicate that the phenomenon is based on an electrophilic attack on DNA. In that respect this phenomenon is different from the basis of the induction of SCE in cultured cells or of

micronuclei in the bone marrow of rodents. The latter two phenomena involve other targets as well. Based on the recognition that the vast majority of recognized human carcinogens are genotoxic, the present finding indicates that the in vivo induction of SCE is a good biomarker, possibly even a biodosimeter, for exposure to potential carcinogens.

CC 4-6 (Toxicology)
Section cross-reference(s): 1

IT 50-07-7, Mitomycin c 50-14-6, Vitamin d2 50-18-0, Cyclophosphamide 50-21-5, biological studies 50-69-1, Ribose 50-71-5, Alloxan 50-76-0, Actinomycin d 51-12-7, Nialamide 51-35-4, Hydroxyproline 51-71-8, Phenelzine 51-79-6, Urethan 52-90-4, Cysteine, biological studies 53-96-3 54-92-2, Iproniazid 55-18-5, Diethylnitrosamine 55-80-1, 3'-Methyl-4-(dimethylamino)azobenzene 56-40-6, Glycine, biological studies 56-41-7, Alanine, biological studies 56-45-1, Serine, biological studies 56-53-1, Diethylstilbestrol 56-81-5, 1,2,3-Propanetriol, biological studies 56-84-8, Aspartic acid, biological studies 56-85-9, Glutamine, biological studies 56-86-0, L-Glutamic acid, biological studies 56-87-1, L-Lysine, biological studies 57-00-1, Creatine 57-10-3, Hexadecanoic acid, biological studies 57-11-4, Octadecanoic acid, biological studies 57-13-6, Urea, biological studies 57-41-0, Phenytoin 57-48-7, Fructose, biological studies 57-50-1, biological studies 57-87-4, Ergosterol 57-88-5, Cholesterol, biological studies 58-08-2, Caffeine, biological studies 58-56-0, Pyridoxine hydrochloride 58-85-5, Biotin 59-30-3, Folic acid, biological studies 59-43-8, Thiamine, biological studies 59-63-2, Isocarboxazid 59-67-6, Niacin, biological studies 59-89-2, N-Nitrosomorpholine 60-09-3, 4-Aminoazobenzene 60-11-7, p-Dimethylaminoazobenzene 60-18-4, Tyrosine, biological studies 60-27-5, Creatinine 60-33-3, 9,12-Octadecadienoic acid (Z,Z)-, biological studies 61-90-5, L-Leucine, biological studies 62-49-7, Choline 62-53-3, Benzenamine, biological studies 62-73-7, Dichlorvos 62-75-9, Dmn 63-42-3, Lactose 63-68-3, Methionine, biological studies 63-91-2, L-Phenylalanine, biological studies 64-17-5, Ethanol, biological studies 64-19-7, Acetic acid, biological studies 64-77-7, Tolbutamide 65-71-4, Thymine 65-86-1, Orotic acid 66-22-8, Uracil, biological studies 67-03-8, Thiamin chloride 67-20-9, Nitrofurantoin 67-66-3, Chloroform, biological studies 67-97-0, Vitamin d3 69-65-8, Mannitol 69-89-6, Xanthine 69-93-2, Uric acid, biological studies 70-18-8, Glutathione, biological studies 70-25-7, Mnng 70-26-8, Ornithine 71-00-1, Histidine, biological studies 71-43-2, Benzene, biological studies 71-44-3, Spermine 72-18-4, Valine, biological studies 72-19-5, L-Threonine, biological studies 73-22-3, Tryptophan, biological studies 73-22-3D, Tryptophan, pyrolyzates, biological studies 73-24-5, Adenine, biological studies 73-32-5, Isoleucine, biological studies 73-40-5, Guanine 74-79-3, Arginine, biological studies 75-25-2, Bromoform 75-27-4, Bromodichloromethane 77-92-9, biological studies 79-83-4, Pantothenic acid 83-88-5, Riboflavine, biological studies 85-87-0, Pyridoxamine 86-54-4, Hydralazine 87-69-4, Tartaric acid, biological studies 87-79-6, Sorbose 87-89-8, myo-Inositol 91-22-5, Quinoline, biological studies 91-59-8, 2-Naphthylamine 94-20-2, Chlorpropamide 95-43-2, D-Threose 95-80-7, 2,4-Diaminotoluene 97-56-3, o-Aminoazotoluene 97-59-6, Allantoin 98-92-0, Niacinamide 100-42-5, biological studies 100-75-4, N-Nitrosopiperidine 101-77-9 106-60-5, δ-Aminolevulinic acid 106-93-4, 1,2-Dibromoethane 107-13-1, 2-Propenenitrile, biological studies 107-35-7, Taurine 109-52-4, n-Valeric acid, biological studies 110-15-6, Butanedioic acid, biological studies 110-17-8, 2-Butenedioic acid (E)-, biological studies 110-60-1, Putrescine 120-62-7, Sulfoxide 124-07-2, Caprylic acid, biological studies 124-48-1, Chlorodibromomethane

126-07-8, Griseofulvin 127-17-3, Pyruvic acid, biological studies 130-89-2, Quinine hydrochloride 134-03-2, Sodium ascorbate 134-32-7, 1-Naphthylamine 137-08-6, Calcium pantothenate 147-85-3, Proline, biological studies 150-13-0, p-Aminobenzoic acid 156-06-9, Phenylpyruvic acid 206-44-0, Fluoranthene 303-45-7, Gossypol 305-84-0, L-Carnosine 327-57-1, Norleucine 328-42-7, Oxaloacetic acid 366-70-1, Natulan 372-75-8, Citrulline 451-13-8, Homogentisic acid 484-23-1, Dihydralazine 488-41-5, Dibromomannitol 512-69-6, Raffinose 522-40-7, Diethylstilbestrol diphosphate 526-95-4, Gluconic acid 541-15-1 541-50-4, Acetoacetic acid, biological studies 589-41-3, N-Hydroxyurethane 615-05-4, 2,4-Diaminoanisole 621-64-7, Dipropylnitrosamine 684-93-5, 1-Methyl-1-nitrosourea 759-73-9, N-Nitroso-N-ethylurea 924-16-3, Dibutylnitrosamine 930-22-3, Butadiene monoepoxide 930-55-2, 1-Nitrosopyrrolidine 951-77-9, Deoxycytidine 951-78-0, Deoxyuridine 964-26-1, Dump 1162-65-8, Aflatoxin b1 1464-53-5, 1,2:3,4-Diepoxybutane 1746-77-6, Isopropylcarbamate 2114-11-6, Allylcarbamate 3416-24-8, Glucosamine 3761-53-3, Ponceau r 3930-19-6, Bruneomycin 4618-18-2, Lactulose 6098-44-8, N-Acetoxy-2-acetylaminofluorene 7235-40-7, β-Carotene 10048-13-2, Sterigmatocystin 11056-06-7, Bleomycin 11103-57-4, Vitamin a 15805-73-9, Vinyl carbamate 18883-66-4, Streptozotocin 20830-75-5, Digoxin 20830-81-3, Daunomycin 25316-40-9, Adriamycin 39715-02-1, Endralazine 52225-20-4, DL-α-Tocopheryl acetate
RL: ADV (Adverse effect, including toxicity); PRP (Properties); BIOL (Biological study)
(dichotomous relationship between DNA reactivity and induction of sister chromatid exchanges in vivo and in vitro)

L92 ANSWER 9 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1992:646681 HCAPLUS Full-text
DOCUMENT NUMBER: 117:246681
TITLE: Characterization of amines by Fast Black K salt in thin-layer chromatography
AUTHOR(S): Ojanpera, Ilkka; Wahala, Kristiina; Hase, Tapio A.
CORPORATE SOURCE: Dep. Forensic Med., Univ. Helsinki, Helsinki, SF-00300, Finland
SOURCE: Analyst (Cambridge, United Kingdom) (1992), 117(10), 1559-65
CODEN: ANALAO; ISSN: 0003-2654
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 26 Dec 1992
AB Amines were characterized on a silica gel TLC plate with the diazonium reagent Fast Black K salt (FBK) and with subsequent novel procedures: acid treatment or treatment with N-(1- naphthyl)ethylenediamine in acid solution The differentiation of primary, secondary, and tertiary aliphatic and aromatic amines was demonstrated, with special attention to drug substances. By using the N-(1- naphthyl)ethylenediamine treatment, a 5-fold improvement in the detection limits for aliphatic secondary amines was achieved compared with FBK alone, allowing detection of 0.01 μg of methamphetamine and 0.04 μg of Me phenidate. The structures of the colored reaction products were elucidated by spectroscopic and TLC methods. An unexpected reaction was observed with dialkylanilines, which reacted by N-coupling with various diazonium salts with cleavage of an alkyl group.
CC 4-2 (Toxicology)
Section cross-reference(s): 1, 25
IT 51-05-8, Procaine hydrochloride 51-57-0, Methamphetamine hydrochloride 60-13-9, Amphetamine sulfate 62-53-3, Aniline, analysis 71-44-3, Spermine 88-05-1, 2,4,6-Trimethylaniline 90-04-0, o-Anisidine

91-66-7, N,N-Diethylaniline 94-09-7, Ethyl 4-aminobenzoate
95-68-1, 2,4-Dimethylaniline 99-97-8, N,N-Dimethyl-p-toluidine
100-01-6, 4-Nitroaniline, analysis 102-27-2, N-Ethyl-m-toluidine
103-69-5, N-Ethylaniline 104-94-9, p-Anisidine 106-49-0, p-Toluidine,
analysis 108-44-1, m-Toluidine, analysis 121-69-7,
N,N-Dimethylaniline, analysis 124-20-9, Spermidine 136-47-0,
Amethocaine hydrochloride 150-13-0, 4-Aminobenzoic acid 156-28-5,
2-Phenylethylamine hydrochloride 333-93-7, Putrescine
hydrochloride 339-43-5, Carbutamide 538-02-3, Cyclopentamine
hydrochloride 557-66-4, Ethylamine hydrochloride 589-08-2,
N-Methyl-2-phenylethylamine 613-97-8, N-Ethyl-N-methylaniline
614-39-1, Procainamide hydrochloride 622-57-1, N-Ethyl-p-toluidine
660-68-4, Diethylamine hydrochloride 665-66-7, Amantadine
hydrochloride 1197-21-3, Phentermine hydrochloride 1786-81-8
2735-04-8, 2,4-Dimethoxyaniline 3665-80-3, N-Ethyl-4-nitroaniline
10541-83-0, N-Methyl-4-aminobenzoic acid 13021-15-3 15467-15-9,
Ethylenediamine hydrochloride 32795-47-4, Nomifensine maleate
56296-78-7, Fluoxetine hydrochloride 71182-65-5 71395-14-7, Tocainide
hydrochloride
RL: ANT (Analyte); ANST (Analytical study)
(thin-layer chromatog. of, Fast Black potassium salt in)

L92 ANSWER 10 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1990:627386 HCAPLUS Full-text
DOCUMENT NUMBER: 113:227386
TITLE: Determination of metabolite and nucleotide concentrations in proliferating lymphocytes by proton NMR of acid extracts
AUTHOR(S): Sze, Daniel Y.; Jardetzky, Oleg
CORPORATE SOURCE: Stanford Magn. Reson. Lab., Stanford Univ., Stanford, CA, 94305-5055, USA
SOURCE: Biochimica et Biophysica Acta, Molecular Cell Research (1990), 1054(2), 181-97
CODEN: BBAMCO; ISSN: 0167-4889
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 22 Dec 1990
AB The major advantages of in vitro 1H-NMR, namely chemical preservation, simultaneous detection, identification, and quantitation of compds., and sensitivity to a large variety of classes of compds., are employed in this study to characterize the metabolic course of mitogen-stimulated proliferation of human peripheral lymphocytes. A reliable method to quantitate amino acids, metabolic intermediates, soluble membrane lipid precursors, and purine, pyridine, and pyrimidine nucleotides is presented, using samples as small as 30 mg wet weight A total of 53 substances were detected in lymphocytes and other blood cells. During the course of lymphocyte culture, changes in intracellular concns. of lactate, taurine, inositol, and nucleotides, including NAD, IMP, and high-energy phosphates, were especially marked. 1H-NMR compares favorably to 31P-NMR and to HPLC, and is especially attractive in light of expectations for future in vivo application.
CC 9-5 (Biochemical Methods)
IT 50-21-5, analysis 50-99-7, Glucose, analysis 51-35-4, Hydroxyproline
53-59-8, NADP 53-84-9, NAD 56-40-6, Glycine, analysis 56-41-7
, Alanine, analysis 56-45-1, Serine, analysis 56-65-5, ATP,
analysis 56-84-8, L-Aspartic acid, analysis 56-85-9,
Glutamine, analysis 56-86-0, L-Glutamic acid, analysis
56-87-1, Lysine, analysis 57-00-1, Creatine 58-61-7,
Adenosine, analysis 58-64-0, ADP, analysis 58-98-0, UDP, analysis
60-00-4, EDTA, analysis 60-18-4, Tyrosine, analysis 61-19-8, AMP,
analysis 61-90-5, Leucine, analysis 62-49-7, Choline 63-39-8, UTP

63-91-2, Phenylalanine, analysis 64-18-6, Formic acid, analysis 64-19-7, Acetic acid, analysis 67-07-2, Phosphocreatine 68-94-0, Hypoxanthine 70-47-3, Asparagine, analysis 71-00-1, Histidine, analysis 71-44-3, Spermine 72-18-4, Valine, analysis 72-19-5, Threonine, analysis 73-32-5, Isoleucine, analysis 85-32-5, GMP 86-01-1, GTP 87-89-8, Inositol 98-92-0, Nicotinamide 107-35-7, Taurine 107-73-3, Phosphorylcholine 107-95-9, β-Alanine 110-15-6, Butanedioic acid, analysis 110-17-8, 2-Butenedioic acid (E)-, analysis 110-60-1, Putrescine 117-96-4, Diatrizoate 124-20-9, Spermidine 127-17-3, analysis 131-99-7, IMP 133-89-1, Uridine diphosphoglucose 138-81-8 146-91-8, GDP 147-85-3, Proline, analysis 497-30-3, Ergothioneine 563-24-6, Glycerophosphorylcholine 1071-23-4, Phosphorylethanolamine 6915-15-7 7365-45-9, Hepes 29908-03-0, S-Adenosyl methionine
RL: ANT (Analyte); ANST (Analytical study)
(determination of, in erythrocytes and neutrophils and proliferating lymphocytes of humans by proton NMR)

L92 ANSWER 11 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1986:48852 HCAPLUS Full-text
DOCUMENT NUMBER: 104:48852
TITLE: Effect of polyamines and guanidines on the growth, nitrogen assimilation and reserve mobilization in germinating radish seeds
AUTHOR(S): Srivastava, S. K.; Kansara, M. S.; Mungre, S. M.
CORPORATE SOURCE: Biochem. Dep., M. S. Univ. Baroda, Baroda, 39002, India
SOURCE: Plant Growth Regulation (1985), 3(3-4), 339-51
CODEN: PGRED3; ISSN: 0167-6903
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 23 Feb 1986
AB Polyamines and guanidines enhanced the growth of radish seedlings grown in dark or light, irresp. of the supply of N. All the compds. inhibited nitrate reductase and glutamine synthetase in the cotyledons of light-grown but not in dark-grown seeds. Nitrite reductase and glutamate dehydrogenase were not affected. Protease was enhanced by all the compds. in dark- as well as in light-grown seeds. Alanine aminotransferase was increased only in the light-grown seeds. The inhibition of nitrate reductase was due not to decreased nitrate uptake, but to a decreased metabolic pool of nitrate and a decline in enzyme synthesis. The inhibition of glutamine synthetase and activation of alanine aminotransferase by the compds. was found only in the chloroplast fraction. The activation of protease was due to the release or activation of preexisting enzyme, whereas that of alanine aminotransferase was dependent on de novo protein synthesis which was abolished by cycloheximide.
CC 11-3 (Plant Biochemistry)
IT 57-00-1 110-60-1 124-20-9
RL: BIOL (Biological study)
(radish embryo growth during germination response to)

L92 ANSWER 12 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1959:123460 HCAPLUS Full-text
DOCUMENT NUMBER: 53:123460
ORIGINAL REFERENCE NO.: 53:22261g-i
TITLE: Releasing chromosome mutations in Vicia faba by the use of cadaverine- and putrescine-hydrochlorides
AUTHOR(S): Rieger, R.; Michaelis, A.
CORPORATE SOURCE: Inst. Cultivated Plants Research, Gattersleben,

Germany
SOURCE: Monatsberichte der Deutschen Akademie der Wissenschaften zu Berlin (1959), 1, 51-3
CODEN: MDAWAH; ISSN: 0011-9814
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
ED Entered STN: 22 Apr 2001
AB On the basis of other work in which mutants were obtained, the authors attempted to bring about chromosomal mutations in Vicia faba by treating the plant with varying concentrations of cadaverine HCl and putrescine HCl. The results were negative. Further study indicated that each of the N-containing agents were decomposed by the action of enzymes in the roots and were not available for activity in the bud.
CC 11D (Biological Chemistry: Botany)

L92 ANSWER 13 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1950:30297 HCAPLUS
DOCUMENT NUMBER: 44:30297
ORIGINAL REFERENCE NO.: 44:5915a-g
TITLE: Raw materials from furfural for polyurethan resins
INVENTOR(S): Codignola, Franco; Piacenza, Mario
DOCUMENT TYPE: Patent
LANGUAGE: Unavailable
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
IT 439947		19481004	IT	<--

ED Entered STN: 22 Apr 2001
AB Furfural 2 mols. is hydrogenated to the alc. at 150° and 130 atmospheric pressure in 20 min. with 10 g. Cu chromite. Tetrahydrofurfuryl alc. is formed at 120° and 90 atmospheric pressure in 80 min. with 8 g. Raney Ni. Dihydropyran is formed by pyrolysis in an alumina column at 380-400° and the fraction b760 86° separated Dihydropyran 6 mols. refluxed with 2 l. 0.2 N HCl for 65 min., neutralized to phenolphthalein with 0.4 N NaOH, and distilled yields 390 g. HO(CH2)4CHO, b3 54-5°; this (2 mols.) is hydrogenated in 750 ml. EtOH at 150° and 150 atmospheric for 15 min. with 10 g. Cu chromite, and the HO(CH2)5OH separated by fractional distillation Alternatively the dihydropyran can be hydrogenated at 90° and 50 atmospheric in 55 min. with 8 g. Raney Ni/mol. to yield tetrahydropyran, b760 88-90°. Treatment for 3 hrs. at 120° with anhydrous HCl and anhydrous chlorides (Ca, Fe, Bi, or Zn) yields Cl(CH2)5Cl, b14 67-8°. Furfural 1 mol. with 41 g. NaOH in 5 l. H2O is oxidized with O in the presence of MnO2 activated with 1% Ag2O, acidified with 20% H2SO4, and the pyromucic acid separated by crystallization The latter (500 g.) pyrolyzed in 500 ml. quinoline with 3 g. MnO2 at 220-5° yields CO2 and furan which is adsorbed in active charcoal, recovered, and converted to tetrahydrofuran at 100-50° and 100-50 atmospheric in 10 min. with 25 g. Raney Ni/500 g. Treated as above, it yields Cl(CH2)4Cl, b12 53-4°. This (500 g.) treated with aqueous concentrated NH3 in the presence of 40 g. Cu oxide and NH4ClO3 at 110° yields H2N(CH2)4NH2.HCl which can then be treated with COCl2. Alternatively the Cl(CH2)4Cl may be treated with 10% NaOH at 10 atmospheric to yield HO(CH2)4OH, b760 230-1°. The HO(CH2)4CHO may be hydrogenated with 30 g. Raney Ni in 1000 g. liquid NH3 at 90 ° and 250 atmospheric for 5 hrs. to yield a compound b1-2 85-90°. To 400 g. of this in 1600 ml. anhydrous alc. is added 400 g. H2SO4 in 1600 ml. anhydrous alc. with cooling; addition of 1600 ml. ether and cooling yields the bisulfate of H2N(CH2)5OH, m. 103°. Treatment with caustic and ether extraction yield the aminopentanol, m. 37 °, which with SOCl2 in HCl yields, upon neutralization and extraction with ether, H2N(CH2)5Cl. Tetrahydrofuran may be prepared from furoic acid by treating 400

g. in 500 ml. 95% alc., with 20 g. Raney Ni and H under 55 atmospheric pressure at 110° for 1 hr. to yield tetrahydrofuroic acid, m. 21°, which is decarboxylated thermally. Alternatively furan is hydrogenated in 15 min. in the presence of 4% Raney Ni and H at 150 atmospheric and 120°. Br(CH2)4Br prepared as above may be treated for 3 hrs. with 2 mols. KCN, extracted with AcOEt, and the NC(CH2)4CN recovered by distillation This (500 g.) is hydrogenated at 130° and 160 atmospheric in 4 parts liquid NH3 with 4% Raney Ni for 3 hrs. and the H2N(CH2)6CN separated Various combinations of these reactions are claimed. Cf. C.A. 43, 4511g.

CC 10 (Organic Chemistry)

IT 97-99-4P, Furfuryl alcohol, tetrahydro- 109-99-9P, Furan, tetrahydro- 110-52-1P, Butane, 1,4-dibromo- 110-56-5P, Butane, 1,4-dichloro- 110-87-2P, Pyran, dihydro- 142-68-7P, Pyran, tetrahydro- 333-93-7P, Putrescine, hydrochloride 628-76-2P, Pentane, 1,5-dichloro- 927-93-5P, 1-Pentanol, 5-amino-, bisulfate 2508-29-4P, 1-Pentanol, 5-amino- 4221-03-8P, Valeraldehyde, 5-hydroxy- 16874-33-2P, 2-Furoic acid, tetrahydro- 23181-80-8P, Heptanenitrile, 7-amino- 59801-88-6P, Pentylamine, 5-chloro-
RL: PREP (Preparation)
(preparation of)

L92 ANSWER 14 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1932:49148 HCAPLUS Full-text
DOCUMENT NUMBER: 26:49148
ORIGINAL REFERENCE NO.: 26:5070i,5071a-b
TITLE: The decomposition of ruflanates, flavianates, picrates and picrolonates by means of wool
AUTHOR(S): Muller, Hellmut
SOURCE: Z. physiol. Chem. (1932), 209, 207-10
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
ED Entered STN: 16 Dec 2001

AB The liberation of organic bases from their insol. salts with various precipitants may be accomplished by adsorption of the precipitant on lamb's wool, preferably in the presence of 0.1 N HCl. The base is then obtained as HCl salt by evaporating the filtrate. One g. of wool adsorbs 0.14-0.19 g. of the precipitant in 48 hrs. at 37°. When larger quantities of precipitated bases are used the bulk of the precipitant may first be removed by Ba(OH)2 treatment before the wool is added. The adsorption treatment is especially useful with bases that are unstable to alkali or oxidation. Examples given are the preparation of betaine-HCl and putrescine-HCl from the rufianates, guanidine-HCl, l-histidine and Me2NH.HCl from the flavianates, betaine-HCl and glycine from the picrates and Me3N.HCl from the picrolonate. The yields were 80-97%.

CC 10 (Organic Chemistry)

IT 50-01-1P, Guanidine, hydrochloride 56-40-6P, Glycine 71-00-1P, Histidine, L- 333-93-7P, Putrescine, hydrochloride 506-59-2P, Dimethylamine, hydrochloride 590-46-5P, Betaine, hydrochloride 593-81-7P, Trimethylamine, hydrochloride
RL: PREP (Preparation)
(preparation of)

=> d L92 15-36 ibib ab hit

L92 ANSWER 15 OF 36 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 94173430 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 8127445
TITLE: High-resolution 1H NMR spectroscopy of cerebrospinal fluid in spinal diseases.

AUTHOR: Koschorek F; Offermann W; Stelten J; Braunsdorf W E; Steller U; Gremmel H; Leibfritz D
CORPORATE SOURCE: University Clinic of Radiology, Kiel, Fed. Rep. of Germany.
SOURCE: Neurosurgical review, (1993) Vol. 16, No. 4, pp. 307-15.
Journal code: 7908181. ISSN: 0344-5607.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199404
ENTRY DATE: Entered STN: 20 Apr 1994
Last Updated on STN: 20 Apr 1994
Entered Medline: 13 Apr 1994

AB Twenty-nine patients with disk herniations, 7 patients with intraspinal tumors, 4 patients with multiple sclerosis and one patient with infection by borrelia have been studied by CT, myelography and/or MR. To gain information on the metabolism of central nervous system disease (CNS), and thus, to improve diagnosis the cerebrospinal fluid (CSF) was studied in all cases using high-resolution 1H NMR spectroscopy at 360 MHz. Seventeen metabolites could be identified in CSF in addition to the usual clinical chemical parameters. As compared to a control group discrimination of tumors from inflammation was possible by means of different metabolites and/or metabolite concentration. The CSF in disk herniations differed in the concentration of acetate from the control group. In CSF of tumors, multiple sclerosis and of infection by borrelia distinct differences in the concentrations of putrescine, citrate, valine, alpha- alanine, acetate, creatinine, glucose, beta-hydroxy-butyric acid, glutamine and creatine have been observed both as compared directly and in comparison to the control group. Thus, high-resolution 1H NMR spectroscopy of CSF gives speedy information on metabolism, since a variety of metabolites, usually examined only in different tests, can be studied in one single step. Thus, high-resolution 1H NMR spectroscopy supports imaging, especially MR, as morphological changes in diseases may be differentiated by means of different metabolite profiles. This assumption needs further confirmation on a prospective study with a larger patient population.

SO Neurosurgical review, (1993) Vol. 16, No. 4, pp. 307-15.
Journal code: 7908181. ISSN: 0344-5607.
AB Twenty-nine patients with disk herniations, 7 patients with intraspinal tumors, 4 patients with multiple sclerosis and one patient with infection by borrelia have been studied by CT, myelography and/or MR. To gain information on the metabolism of central nervous system disease (CNS), and thus, to improve diagnosis the cerebrospinal fluid (CSF) was studied in all cases using high-resolution 1H NMR spectroscopy at 360 MHz. Seventeen metabolites could be identified in CSF in addition to the usual clinical chemical parameters. As compared to a control group discrimination of tumors from inflammation was possible by means of different metabolites and/or metabolite concentration. The CSF in disk herniations differed in the concentration of acetate from the control group. In CSF of tumors, multiple sclerosis and of infection by borrelia distinct differences in the concentrations of putrescine, citrate, valine, alpha- alanine, acetate, creatinine, glucose, beta-hydroxy-butyric acid, glutamine and creatine have been observed both as compared directly and in comparison to the control group. Thus, high-resolution 1H NMR spectroscopy of CSF gives speedy information on metabolism, since a variety of metabolites, usually examined only in different tests, can be studied in one single step. Thus, high-resolution 1H NMR spectroscopy supports imaging, especially MR, as morphological changes in diseases may be differentiated by means of different metabolite profiles. This assumption needs further confirmation on a prospective study with a larger patient population.

L92 ANSWER 16 OF 36 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 83072836 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 6816107
TITLE: alpha-Difluoromethylornithine: a promising lead for preventive chemotherapy for coccidiosis.
AUTHOR: Hanson W L; Bradford M M; Chapman W L Jr; Waits V B; McCann P P; Sjoerdsma A
SOURCE: American journal of veterinary research, (1982 Sep) Vol. 43, No. 9, pp. 1651-3.
Journal code: 0375011. ISSN: 0002-9645.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198301
ENTRY DATE: Entered STN: 17 Mar 1990
Last Updated on STN: 17 Mar 1990
Entered Medline: 19 Jan 1983

AB alpha-Difluoromethylornithine (DFMO; RMI 71,782) given in drinking water in concentrations as low as 0.0625% inhibited infections of Eimeria tenella and minimized the development of lesions in chickens. It had approximately the same activity as a currently used anticoccidial drug, amprolium, and also had the advantage of being relatively nontoxic in chickens. Body weight gains were not reduced in chickens given 0.0635% DFMO or less for 14 days starting 8 days before they were inoculated with oocysts, but were reduced in chickens given drinking water containing 0.125 and 0.25% DFMO. The anticoccidial activity of DFMO was completely reversed by injection (intraabdominal) of putrescine hydrochloride (300 mg/kg of body weight/day), indicating that the drug may act by blocking putrescine biosynthesis. Inoculated chickens, in which coccidial lesion development was suppressed by DFMO, resisted subsequent challenge exposure with E tenella, as did nontreated infected control birds which had recovered from infection.

SO American journal of veterinary research, (1982 Sep) Vol. 43, No. 9, pp. 1651-3.
Journal code: 0375011. ISSN: 0002-9645.

AB alpha-Difluoromethylornithine (DFMO; RMI 71,782) given in drinking water in concentrations as low as 0.0625% inhibited infections of Eimeria tenella and minimized the development of lesions in chickens. It had approximately the same activity as a currently used anticoccidial drug, amprolium, and also had the advantage of being relatively nontoxic in chickens. Body weight gains were not reduced in chickens given 0.0635% DFMO or less for 14 days starting 8 days before they were inoculated with oocysts, but were reduced in chickens given drinking water containing 0.125 and 0.25% DFMO. The anticoccidial activity of DFMO was completely reversed by injection (intraabdominal) of putrescine hydrochloride (300 mg/kg of body weight/day), indicating that the drug may act by blocking putrescine biosynthesis. Inoculated chickens, in which coccidial lesion development was suppressed by DFMO, resisted subsequent challenge exposure with E tenella, as did nontreated infected control birds which had recovered from infection.

CT Check Tags: Male
Animals
Cecal Diseases: PC, prevention & control
Cecal Diseases: VE, veterinary
*Chickens: PS, parasitology
Coccidiosis: PC, prevention & control
*Coccidiosis: VE, veterinary
Coccidiostats: AI, antagonists & inhibitors
*Coccidiostats: TU, therapeutic use
Eflornithine
Eimeria: DE, drug effects

*Ornithine: AA, analogs & derivatives
Ornithine: AI, antagonists & inhibitors
Ornithine: PD, pharmacology
Ornithine: TU, therapeutic use
*Poultry Diseases: PC, prevention & control
Putrescine: PD, pharmacology

L92 ANSWER 17 OF 36 MEDLINE on STN
ACCESSION NUMBER: 94362517 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 8081216
TITLE: Modification of pig kidney diamine oxidase with ethoxyformic anhydride and rose bengal: evidence for essential histidyl residue at the active site.
AUTHOR: Shah M A; Ali R
CORPORATE SOURCE: Department of Biochemistry, Faculty of Medicine, A.M.U. Aligarh, India.
SOURCE: Biochemistry and molecular biology international, (1994 May) Vol. 33, No. 1, pp. 9-19.
Journal code: 9306673. ISSN: 1039-9712.
PUB. COUNTRY: Australia
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199410
ENTRY DATE: Entered STN: 21 Oct 1994
Last Updated on STN: 6 Feb 1998
Entered Medline: 11 Oct 1994

AB Purified diamine oxidase from pig kidney showed time dependent inactivation by ethoxyformic anhydride and photooxidation with Rose Bengal. Modification of histidine either by ethoxyformic anhydride or by Rose Bengal was the sole cause of enzyme inactivation. The inactivated enzyme showed no significant perturbation in structure. The protection against photooxidation of enzymatic activity and histidine residues by inhibitor (phenylenediamine hydrochloride) and substrate (putrescine) protected the photooxidation of two histidyl residues. However, kinetic analysis of photooxidation of histidyl residues and inactivation of the enzyme conclusively suggested the involvement of one histidyl residue, which seems to be located at the active site of diamine oxidase.

SO Biochemistry and molecular biology international, (1994 May) Vol. 33, No. 1, pp. 9-19.
Journal code: 9306673. ISSN: 1039-9712.

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CT *Amine Oxidase (Copper-Containing): AI, antagonists & inhibitors
Amine Oxidase (Copper-Containing): CH, chemistry
Animals
Binding Sites
*Diethyl Pyrocarbonate: PD, pharmacology
*Histidine: AN, analysis
Hydroxylamine

Hydroxylamines: PD, pharmacology
Kidney: DE, drug effects
*Kidney: EN, enzymology
Kinetics
Oxidants, Photochemical: CH, chemistry
Oxidants, Photochemical: PD, pharmacology
Putrescine: CH, chemistry
Rose Bengal: PD, pharmacology
Spectrometry, Fluorescence
Substrate Specificity
Swine

RN 110-60-1 (Putrescine); 11121-48-5 (Rose Bengal); 1609-47-8 (Diethyl Pyrocarbonate); 71-00-1 (Histidine); 7803-49-8 (Hydroxylamine)

L92 ANSWER 18 OF 36 MEDLINE on STN
ACCESSION NUMBER: 91178507 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 2079625
TITLE: Comparative studies on the degradation of guanidino and ureido compounds by Pseudomonas.
AUTHOR: Tricot C; Pierard A; Stalon V
CORPORATE SOURCE: Laboratoire de Microbiologie, Faculte des Sciences, Universite Libre de Bruxelles, Belgium.
SOURCE: Journal of general microbiology, (1990 Nov) Vol. 136, No. 11, pp. 2307-17.
Journal code: 0375371. ISSN: 0022-1287.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199105
ENTRY DATE: Entered STN: 19 May 1991
Last Updated on STN: 19 May 1991
Entered Medline: 1 May 1991

AB The utilization of guanidino and ureido compounds was studied in several Pseudomonas species. Multiple routes of agmatine catabolism were found. All members of the homology group I of Pseudomonas use the initial deamination of agmatine to carbamoylputrescine which is subsequently converted to putrescine. In Pseudomonas indigofera, the catabolism of agmatine can also occur via an initial hydrolysis of the amidino group to putrescine catalyzed by an agmatine amidinohydrolase. A third pathway was found in Pseudomonas cepacia, namely oxidative deamination producing guanidinobutyraldehyde catalyzed by agmatine dehydrogenase, followed by formation of guanidinobutyrate and removal of urea by guanidinobutyrate amidinohydrolase to produce 4-aminobutyrate. Novel amidino-hydrolases were characterized in P. putida for the utilization of arcaine and audouine, and in P. cepacia for arcaine, homoarginine and guanidinovalerate. Guanidinovalerate amidinohydrolase was also detected in P. doudoroffii. Some of these amidinohydrolases accept more than one substrate, e.g., guanidinobutyrate and guanidinovalerate utilization by P. doudoroffii and P. cepacia, the catabolism of arcaine and audouine by P. putida, and the degradation of arcaine and homoarginine by P. cepacia.

TI Comparative studies on the degradation of guanidino and ureido compounds by Pseudomonas.

SO Journal of general microbiology, (1990 Nov) Vol. 136, No. 11, pp. 2307-17.
Journal code: 0375371. ISSN: 0022-1287.

AB The utilization of guanidino and ureido compounds was studied in several Pseudomonas species. Multiple routes of agmatine catabolism were found. All members of the homology group I of Pseudomonas use the initial deamination of

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CT Amidohydrolases: ME, metabolism
Creatine: ME, metabolism
Creatinine: ME, metabolism
*Guanidines: ME, metabolism
Hydrolysis
Pseudomonas: EN, enzymology
Pseudomonas: GD, growth & development
*Pseudomonas: ME, metabolism
*Urea: AA, analogs & derivatives
Urea: ME, metabolism

RN 57-00-1 (Creatine); 57-13-6 (Urea); 60-27-5 (Creatinine)

CN 0 (Guanidines); EC 3.5.- (Amidohydrolases); EC 3.5.2.10 (creatininase)

L92 ANSWER 19 OF 36 MEDLINE on STN

ACCESSION NUMBER: 85260587 MEDLINE Full-text

DOCUMENT NUMBER: PubMed ID: 3926714

TITLE: Excretion of polyamines by humans following inhibition of diamine oxidase.

AUTHOR: Chayen R; Goldberg S; Burke M

SOURCE: Israel journal of medical sciences, (1985 Jun) Vol. 21, No. 6, pp. 543-5.
Journal code: 0013105. ISSN: 0021-2180.

PUB. COUNTRY: Israel

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 198509

ENTRY DATE: Entered STN: 20 Mar 1990
Last Updated on STN: 20 Mar 1990
Entered Medline: 23 Sep 1985

SO Israel journal of medical sciences, (1985 Jun) Vol. 21, No. 6, pp. 543-5.
Journal code: 0013105. ISSN: 0021-2180.

CT Check Tags: Male
*Amine Oxidase (Copper-Containing): AI, antagonists & inhibitors
Cadaverine: UR, urine
Creatine: UR, urine
*Guanidines: PD, pharmacology
Humans
*Polyamines: UR, urine
Putrescine: UR, urine
Spermidine: UR, urine
Spermine: UR, urine

RN 110-60-1 (Putrescine); 124-20-9 (Spermidine); 462-94-2 (Cadaverine); 57-00-1 (Creatine); 71-44-3 (Spermine); 79-17-4

(pimagedine)
CN 0 (Guanidines); 0 (Polyamines); EC 1.4.3.6 (Amine Oxidase (Copper-Containing))

L92 ANSWER 20 OF 36 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 1
ACCESSION NUMBER: 2003:57203 BIOSIS Full-text
DOCUMENT NUMBER: PREV200300057203
TITLE: Human, rat and chicken small intestinal Na+-Cl--creatine transporter: Functional, molecular characterization and localization.
AUTHOR(S): Peral, M. J.; Garcia-Delgado, M.; Calonge, M. L.; Duran, J. M.; De la Horra, M. C.; Wallimann, T.; Speer, O.; Ilundain, A. A. [Reprint Author]
CORPORATE SOURCE: Depto. Fisiologia y Biologia Animal, Facultad de Farmacia, Tramontana s/n, 41012, Sevilla, Spain
ilundain@us.es
SOURCE: Journal of Physiology (Cambridge), (15 November 2002) Vol. 545, No. 1, pp. 133-144. print.
ISSN: 0022-3751 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 22 Jan 2003
Last Updated on STN: 22 Jan 2003
AB In spite of all the fascinating properties of oral creatine supplementation, the mechanism(s) mediating its intestinal absorption has(have) not been investigated. The purpose of this study was to characterize intestinal creatine transport. (14C)Creatine uptake was measured in chicken enterocytes and rat ileum, and expression of the creatine transporter CRT was examined in human, rat and chicken small intestine by reverse transcription-polymerase chain reaction, Northern blot, in situ hybridization, immunoblotting and immunohistochemistry. Results show that enterocytes accumulate creatine against its concentration gradient. This accumulation was electrogenic, Na+- and Cl--dependent, with a probable stoichiometry of 2 Na+: 1 Cl-: 1 creatine, and inhibited by ouabain and iodoacetic acid. The kinetic study revealed a Km for creatine of 29 muM. (14C)Creatine uptake was efficiently antagonized by non-labelled creatine, guanidinopropionic acid and cyclocreatine. More distant structural analogues of creatine, such as GABA, choline, glycine, beta-alanine, taurine and betaine, had no effect on intestinal creatine uptake, indicating a high substrate specificity of the creatine transporter. Consistent with these functional data, messenger RNA for CRT was detected only in the cells lining the intestinal villus. The sequences of partial clones, and of the full-length cDNA clone, isolated from human and rat small intestine were identical to previously cloned CRT cDNAs. Immunological analysis revealed that CRT protein was mainly associated with the apical membrane of the enterocytes. This study reports for the first time that mammalian and avian enterocytes express CRT along the villus, where it mediates high-affinity, Na+- and Cl--dependent, apical creatine uptake.
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ISSN: 0022-3751 (ISSN print).
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IT Major Concepts
Biochemistry and Molecular Biophysics; Digestive System (Ingestion and Assimilation)

IT Parts, Structures, & Systems of Organisms
apical membrane; enterocytes: digestive system; ileum: digestive system; small intestine: digestive system

IT Chemicals & Biochemicals
beta-alanine; betaine; chloride ion; choline; creatine: intake; cyclocreatine; gamma-aminobutyric acid; glycine; guanidinopropionic acid; iodoacetic acid; ouabain; small intestinal sodium ion-chloride ion-creatine transporter: function, localization, molecular characterization; small intestinal sodium ion-chloride ion-creatine transporter messenger RNA: expression; sodium ion; taurine

RN 107-95-9 (beta-alanine)
107-43-7 (betaine)
16887-00-6 (chloride ion)
62-49-7 (choline)
57-00-1 (creatine)
35404-50-3 (cyclocreatine)
56-12-2 (gamma-aminobutyric acid)
56-40-6 (glycine)
353-09-3 (guanidinopropionic acid)
64-69-7 (iodoacetic acid)
630-60-4 (ouabain)
17341-25-2 (sodium ion)
107-35-7 (taurine)

L92 ANSWER 21 OF 36 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 2001:370776 BIOSIS Full-text
DOCUMENT NUMBER: PREV200100370776
TITLE: Functional characterization of small intestine creatine transport.
AUTHOR(S): Ilundain, A. A. [Reprint author]; Garcia-Delgado, M. [Reprint author]; Peral, M. J. [Reprint author]; Duran, J. M. [Reprint author]; Calonge, M. L. [Reprint author]
CORPORATE SOURCE: Departamento Fisiologia y Biologia Animal, Universidad de Sevilla, 41012, Sevilla, Spain
SOURCE: Journal of Physiology (Cambridge), (May, 2001) Vol. 533P, pp. 63P. print.
Meeting Info.: Proceedings of the Scientific Meeting of The Physiological Society. Oxford, England, UK. March 19-21, 2001. Physiological Society.
CODEN: JPHYA7. ISSN: 0022-3751.

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 8 Aug 2001
Last Updated on STN: 19 Feb 2002
SO Journal of Physiology (Cambridge), (May, 2001) Vol. 533P, pp. 63P. print.
Meeting Info.: Proceedings of the Scientific Meeting of The Physiological Society. Oxford, England, UK. March 19-21, 2001. Physiological Society.
CODEN: JPHYA7. ISSN: 0022-3751.
IT Major Concepts
Biochemistry and Molecular Biophysics; Digestive System (Ingestion and Assimilation)
IT Parts, Structures, & Systems of Organisms
enterocytes: digestive system; small intestine: digestive system
IT Chemicals & Biochemicals
GABA [gamma-aminobutyric acid]; beta-alanine; betaine; choline; creatinine: transport; glycine; guanidinopropionic acid; iodoacetic acid; nipecotic acid; ouabain; sodium chloride; taurine; valinomycin
RN 56-12-2 (GABA)
56-12-2 (gamma-aminobutyric acid)
107-95-9 (beta-alanine)
107-43-7 (betaine)
62-49-7 (choline)
60-27-5 (creatinine)
56-40-6 (glycine)
353-09-3 (guanidinopropionic acid)
64-69-7 (iodoacetic acid)
498-95-3 (nipecotic acid)
630-60-4 (ouabain)
7647-14-5 (sodium chloride)
107-35-7 (taurine)
2001-95-8 (valinomycin)

L92 ANSWER 22 OF 36 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 1984:318296 BIOSIS Full-text
DOCUMENT NUMBER: PREV198478054776; BA78:54776
TITLE: EFFECT OF AMINES AND GUANIDINES ON PEROXIDASE FROM MAIZE ZEA-MAYS SCUTELLUM.
AUTHOR(S): SRIVASTAVA S K [Reprint author]; RAJBABU P
CORPORATE SOURCE: BIOCHEM DEP, MS UNIV BARODA, BARODA 390 002, INDIA
SOURCE: Phytochemistry (Oxford), (1983) Vol. 22, No. 12, pp. 2681-2686.
CODEN: PYTCAS. ISSN: 0031-9422.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
AB The membrane-bound peroxidase activity of excised maize scutellum is inhibited by putrescine, spermidine and spermine and activated by guanidino-acetic acid, guanidino-butyric acid, guazatine and dodine as a result of their binding to the membranes. The inhibition of polyamines is reversed by guanidino compounds but the activation by guanidines is not reversed by polyamines. Other guanidino compounds like arginine, agmatine, creatine and creatinine have no effect by themselves but they reverse the effect of polyamines, except in the case of creatine which does not have a free guanidino group. Peroxidase present in the soluble fraction or the ionically bound peroxidase from particulate fractions solubilized by Ca2+ is not affected by polyamines or

guanidines. The sulfhydryl reagents iodoacetate and p-chloromercuribenzoate (p-CMB) activate peroxidase activity and compete for the polyamine binding site. The effect of dodine is potentiated by sulfhydryl reagents.

TI EFFECT OF AMINES AND GUANIDINES ON PEROXIDASE FROM MAIZE ZEA-MAYS SCUTELLUM.

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RN 113-00-8D (GUANIDINES)
9003-99-0 (PEROXIDASE)
13940-21-1 (SULFHYDRYL)

L92 ANSWER 23 OF 36 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1981:268226 BIOSIS Full-text
DOCUMENT NUMBER: PREV198172053210; BA72:53210
TITLE: URINARY PUTRESCINE AND PLASMA LACTATE DEHYDROGENASE AS MARKERS OF EXPERIMENTAL ADENO CARCINOMA GROWTH.
AUTHOR(S): ANEHUS S [Reprint author]; BENGTSSON G; ANDERSSON G; CARLSSON G; HAFSTROM L; YNGNER T; HEBY O
CORPORATE SOURCE: DEP ZOOPHYSIOL, UNIV LUND, HELGONAVAGEN 3B, S-223 62 LUND, SWED
SOURCE: European Journal of Cancer, (1981) Vol. 17, No. 5, pp. 511-518. CODEN: EJCAAH. ISSN: 0014-2964.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB The objective of this study was to assess, in a controlled experimental system, whether changes in urinary polyamine excretion reflect growth of a solid tumor, and whether such changes are dependent on tumor location. A transplantable N-methyl-N'-nitro-N-nitrosoguanidine-induced adenocarcinoma (NG-W1) was grown intrahepatically or s.c. in male Wistar rats. Tumor size was measured at various time intervals and blood samples and 24 h urines were collected. Analyses of 24 h urines for their polyamine content, using thin-layer chromatography, revealed a positive correlation between the 24 h putrescine output and the increasing tumor burden. The 24 h urine volume paralleled the increase in 24 h putrescine excretion. The 24 h urinary excretion of spermidine remained constant throughout tumor growth, as did that of creatinine. Analyses of blood plasma for its lactate dehydrogenase activity, using a spectrophotometric technique, indicated no relationship between plasma lactate dehydrogenase activity and tumor burden, except at a large tumor mass. The increase in 24 h urinary putrescine excretion in rats harboring an intrahepatic tumor preceded that which occurred in rats harboring a s.c. tumor. This time lapse was attributable to the fact that the tumor growth characteristics, including vascularization, differed between the 2 locations, intrahepatic tumors having more extensive growth and better

vascularization than s.c. tumors. The urine putrescine excretion may be helpful in appraising relapse and recurrence of cancer.

TI URINARY PUTRESCINE AND PLASMA LACTATE DEHYDROGENASE AS MARKERS OF EXPERIMENTAL ADENO CARCINOMA GROWTH.

SO European Journal of Cancer, (1981) Vol. 17, No. 5, pp. 511-518.
CODEN: EJCAAH. ISSN: 0014-2964.

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IT Miscellaneous Descriptors
RAT NG-W-1 CELLS N METHYL-N'-NITRO-N-NITROSO GUANIDINE
CARCINOGEN HEPATIC TUMOR SUB CUTANEOUS TUMOR TUMOR VASCULARIZATION

RN 110-60-1 (PUTRESCINE)
9001-60-9 (LACTATE DEHYDROGENASE)
70-25-7 (N-METHYL-N'-NITRO-N-NITROSOGUANIDINE)

L92 ANSWER 24 OF 36 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1942:21579 BIOSIS Full-text
DOCUMENT NUMBER: PREV19421600021668; BA16:21668
TITLE: Enzymhemmung und Enzymblockierung.
AUTHOR(S): EDLBACHER, S.; BAUR, H.; BECKER, M H.
CORPORATE SOURCE: U. Basel
SOURCE: HOPPE SEYLER S ZEITSCHR PHYSIOL CHEM, (1940) Vol. 265, No. 2/3, pp. 61-71.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable
ENTRY DATE: Entered STN: May 2007
Last Updated on STN: May 2007

AB When a small amount of the natural substrate and a large amount of an altered substrate was added to an enzyme solution, in many cases, any action on the natural substrate was prevented. Histidinase of mammalian liver was very specific, acting only on l-histidine with the formation of NH3. This action was 90% inhibited in the presence of large amts. of the unnatural d-histidine or other imidazoles, such as histamine, imidazole, 1- and 4-imidazolelactic acid and imidazoleacetic acid. Ethylenediamine, lysine, ornithine, guanidine, methylguanidine, dimethylguanidine, octopine, arcaine and creatinine blocked the reaction, but in some cases to a less extent. Argininic acid, cadaverine, putrescine, ethylenetriamine, agmatine, creatine and guanidine acetic acid had little effect. The histidinase formed a compound with the unnatural antipode, and thus the action of the enzyme was blocked. The enzyme solution was

prepared by grinding the livers of 3 rats with quartz sand and 4 vols. of phosphate buffer, pH 8. Histidine hydrochloride was dissolved in H2O, neutralized and the solution adjusted to M/10 concentrate with the phosphate buffer. Solns. of the inhibiting compounds were prepared in the same manner. The reaction mixture contained 3 cc. of the histidine solution, 0.5-12 cc. of the inhibitor solution, and 4 cc. of the enzyme solution The volume was made up to 25 cc. with the buffer and 1 cc. toluol added. After standing about 21 hrs. at 38[degree] in the thermostat, 2 cc. of 30% NaOH were added and the NH3 in N/50 H2SO4 back titrated according to Folin. ABSTRACT AUTHORS: A. B. McCoord

SO HOPPE SEYLER S ZEITSCHR PHYSIOL CHEM, (1940) Vol. 265, No. 2/3, pp. 61-71.

AB When a small amount of the natural substrate and a large amount of an altered substrate was added to an enzyme solution, in many cases, any action on the natural substrate was prevented. Histidinase of mammalian liver was very specific, acting only on l-histidine with the formation of NH3. This action was 90% inhibited in the presence of large amts. of the unnatural d-histidine or other imidazoles, such as histamine, imidazole, 1- and 4-imidazolelactic acid and imidazoleacetic acid. Ethylenediamine, lysine, ornithine, guanidine, methylguanidine, dimethylguanidine, octopine, arcaine and creatinine blocked the reaction, but in some cases to a less extent. Argininic acid, cadaverine, putrescine, ethylenetriamine, agmatine, creatine and guanidine acetic acid had little effect. The histidinase formed a compound with the unnatural antipode, and thus the action of the enzyme was blocked. The enzyme solution was prepared by grinding the livers of 3 rats with quartz sand and 4 vols. of phosphate buffer, pH 8. Histidine hydrochloride was dissolved in H2O, neutralized and the solution adjusted to M/10 concentrate with the phosphate buffer. Solns. of the inhibiting compounds were prepared in the same manner. The reaction mixture contained 3 cc. of the histidine solution, 0.5-12 cc. of the inhibitor solution, and 4 cc. of the enzyme solution The volume was made up to 25 cc. with the buffer and 1 cc. toluol added. After standing about 21 hrs. at 38[degree] in the thermostat, 2 cc. of 30% NaOH were added and the NH3 in N/50 H2SO4 back titrated according to Folin. ABSTRACT AUTHORS: A. B. McCoord

IT Major Concepts
Enzymology (Biochemistry and Molecular Biophysics)

IT Parts, Structures, & Systems of Organisms
liver: digestive system

IT Chemicals & Biochemicals
imidazole; guanidine; methylguanidine; putrescine; Ethylenediamine; lysine; guanidine acetic acid; phosphate buffer; creatine; histamine; creatinine; cadaverine; histidine; histidinase [EC 4.3.1.3]; ornithine; 4-imidazolelactic acid; agmatine; acetic acid; dimethylguanidine; ethylenetriamine; imidazoleacetic acid

RN 288-32-4 (imidazole)
113-00-8 (guanidine)
471-29-4 (methylguanidine)
110-60-1 (putrescine)
70-54-2 (lysine)
352-97-6 (guanidine acetic acid)
57-00-1 (creatine)
51-45-6 (histamine)
60-27-5 (creatinine)
462-94-2 (cadaverine)
4998-57-6 (histidine)
616-07-9 (ornithine)
306-60-5 (agmatine)
64-19-7 (acetic acid)
30581-89-6 (imidazoleacetic acid)

L92 ANSWER 25 OF 36 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 1937:3632 BIOSIS Full-text
DOCUMENT NUMBER: PREV19371100003634; BA11:3634
TITLE: Weitere Beobachtungen an uber-lebenden blutbildenden Organen.
AUTHOR(S): JENEY, A. V.
SOURCE: VIRCHOWS ARCH PATH ANAL U PHYSIOL, (1934) Vol. 293, No. 4, pp. 665-673.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable
ENTRY DATE: Entered STN: May 2007
Last Updated on STN: May 2007

AB Observations were made on the effects of various substances upon erythropoesis in cultures of marrow and splenic tissue in plasma. Prolin and the non-saponifiable part of liver intensified the stimulating effect of arginin on erythropoesis. FeCl2, Cu2Cl2 and COCl2 had the same effect, and this was increased by addition of globin or hematoporphyrin. Irradiation of arginin did not modify its stimulating effect, but treatment with nascent HNO3 decreased it as did also decarboxylization to agmatin. Of substances related to arginin, only guanidin, and to a slight extent cadayerin, had an accelerating effect on erythropoesis. Creatinin, ornithin, putrescin, spermin had no effect, and spermidin almost none. Of poisons, pyro-gallic acid and phenylhydrazine produced an increase in normoblasts and megaloblasts but an increase in normal erythrocytes only when cholesterin was added. Toluylen-diamin resulted in hemolysis and the production of mye-locytes; and benzol and de-oxy-cholic acid resulted in aplasia. Ether extracts of white and yellow waxes, and solutions of carotin caused a marked increase in erythropoesis. Vitamin B accelerated it somewhat less, and vitamin C and D were practically without effect. ABSTRACT AUTHORS: E. H. Tompkins

SO VIRCHOWS ARCH PATH ANAL U PHYSIOL, (1934) Vol. 293, No. 4, pp. 665-673.

AB Observations were made on the effects of various substances upon erythropoesis in cultures of marrow and splenic tissue in plasma. Prolin and the non-saponifiable part of liver intensified the stimulating effect of arginin on erythropoesis. FeCl2, Cu2Cl2 and COCl2 had the same effect, and this was increased by addition of globin or hematoporphyrin. Irradiation of arginin did not modify its stimulating effect, but treatment with nascent HNO3 decreased it as did also decarboxylization to agmatin. Of substances related to arginin, only guanidin, and to a slight extent cadayerin, had an accelerating effect on erythropoesis. Creatinin, ornithin, putrescin, spermin had no effect, and spermidin almost none. Of poisons, pyro-gallic acid and phenylhydrazine produced an increase in normoblasts and megaloblasts but an increase in normal erythrocytes only when cholesterin was added. Toluylen-diamin resulted in hemolysis and the production of mye-locytes; and benzol and de-oxy-cholic acid resulted in aplasia. Ether extracts of white and yellow waxes, and solutions of carotin caused a marked increase in erythropoesis. Vitamin B accelerated it somewhat less, and vitamin C and D were practically without effect. ABSTRACT AUTHORS: E. H. Tompkins

IT Major Concepts
Pharmacology
IT Parts, Structures, & Systems of Organisms
splenic tissue; plasma: blood and lymphatics; liver: digestive system; megaloblasts: blood and lymphatics; erythrocytes: blood and lymphatics; normoblasts
IT Chemicals & Biochemicals
benzol; vitamin C; guanidin; globin; phenylhydrazine; hematoporphyrin

L92 ANSWER 26 OF 36 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 1931:13790 BIOSIS Full-text
DOCUMENT NUMBER: PREV19310500013821; BA05:13821
TITLE: No English Title Available.
Original Title: Die Chini-zarinsulfosaure (Rufiansaure) als Fallungsmittel.
AUTHOR(S): ZIMMERMANN, WALTHER
SOURCE: HOPPE SEYLER S ZEITSCHR PHYSIOL CHEM, (1930) Vol. 188, No. 3/5, pp. 180-188.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable
ENTRY DATE: Entered STN: May 2007
Last Updated on STN: May 2007
AB This compound was found useful in precipitating organic bases and certain of the amino acids. The following compounds could be thrown out of solution by it: creatinine, putrescine, arginine, guanidine, betaine, lysine and choline.
SO HOPPE SEYLER S ZEITSCHR PHYSIOL CHEM, (1930) Vol. 188, No. 3/5, pp. 180-188.
AB This compound was found useful in precipitating organic bases and certain of the amino acids. The following compounds could be thrown out of solution by it: creatinine, putrescine, arginine, guanidine, betaine, lysine and choline.
IT Major Concepts
Physiology
IT Chemicals & Biochemicals
lysine; guanidine; creatinine; arginine; putrescine; amino acids; choline; betaine
RN 70-54-2 (lysine)
113-00-8 (guanidine)
60-27-5 (creatinine)
7200-25-1 (arginine)
110-60-1 (putrescine)
62-49-7 (choline)
107-43-7 (betaine)

L92 ANSWER 27 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 4
ACCESSION NUMBER: 1990123237 EMBASE Full-text
TITLE: Assessment of renal toxicity by urinary enzymes in patients receiving chemotherapy with 8-methyl-8-acetylenic-putrescine.
AUTHOR: Carmichael J.; Cantwell B.M.J.; Harris A.L.; Buamah P.K.; Hodson A.W.; Skillen A.W.
CORPORATE SOURCE: J. Carmichael, ICRF Dept. Clinical Oncology, Churchill Hospital, Headington, OX3 7LJ, United Kingdom
SOURCE: Cancer Chemotherapy and Pharmacology, (1990) Vol. 26, No. 1, pp. 65-66.
ISSN: 0344-5704 CODEN: CCPHDZ
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 13 Dec 1991
Last Updated on STN: 13 Dec 1991

AB Renal toxicity was assessed in 19 patients receiving methyl acetylenic putrescine (MAP), an irreversible inhibitor of ornithine decarboxylase. Patients received 250 mg t. d. s. for up to 13 weeks. This dose effectively inhibited the target enzyme, as shown by elevations in decarboxylated S-adenosyl methionine levels. No significant nephrotoxicity was observed in these patients as determined by plasma urea, creatinine and creatinine clearance measurements, although minor elevations of the urinary enzymes lactate dehydrogenase, N-acetyl-β-glucosaminidase, alkaline phosphatase and alanine aminopeptidase were observed. As this could represent sub-clinical renal damage, caution should be excercised when using MAP in combination with other cytotoxic drugs.

TI Assessment of renal toxicity by urinary enzymes in patients receiving chemotherapy with 8-methyl-8-acetylenic-putrescine.

SO Cancer Chemotherapy and Pharmacology, (1990) Vol. 26, No. 1, pp. 65-66. ISSN: 0344-5704 CODEN: CCPHDZ

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CT Medical Descriptors:
adult
aged
article
clinical article
human
*nephrotoxicity: SI, side effect
oral drug administration
priority journal

CT Drug Descriptors:
*6 heptyne 2,5 diamine: AE, adverse drug reaction
*6 heptyne 2,5 diamine: CT, clinical trial
*6 heptyne 2,5 diamine: DT, drug therapy
*kidney enzyme

L92 ANSWER 28 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2006203613 EMBASE Full-text
TITLE: The role of MRI and PET/SPECT in Alzheimer's disease.
AUTHOR: Coimbra A.; Williams D.S.; Hostetler E.D.
CORPORATE SOURCE: A. Coimbra, Department of Imaging Research, Merck Research Labs., West Point, PA 19486, United States. eric_hostetler@merck.com
SOURCE: Current Topics in Medicinal Chemistry, (Mar 2006) Vol. 6, No. 6, pp. 629-647. Refs: 171 ISSN: 1568-0266 CODEN: CTMCCL
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 014 Radiology
023 Nuclear Medicine
029 Clinical and Experimental Biochemistry
032 Psychiatry
037 Drug Literature Index

008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 18 May 2006
Last Updated on STN: 18 May 2006

AB Alzheimer's disease (AD) is difficult to diagnose in its early stages, and even if detected early, there is no preventative treatment. Imaging modalities such as MRI, PET, and SPECT have the potential to contribute to both the diagnosis of Alzheimer's disease, as well as assist in the search for more effective treatments. A number of AD-related biomarkers have been proposed and evaluated. The use of PET imaging to detect alterations in regional brain metabolism using [(18)F]FDG has enabled more sensitive and accurate early diagnosis of AD, especially in conjunction with traditional medical evaluation. Additionally, magnetic resonance imaging and spectroscopy provide a wide range of biomarkers that have been shown to correlate with the progression of AD. Some of these markers have been pursued in clinical trials. Progress has been made toward the evaluation of other more AD-specific biomarkers. However, many questions remain concerning the validity and sensitivity of these imaging biomarkers, to aid in the assessment of potential new treatments, especially those related to increased levels of amyloid peptides in the brain. .COPYRGT. 2006 Bentham Science Publishers Ltd.

DT Journal; General Review; (Review)

AB Alzheimer's disease (AD) is difficult to diagnose in its early stages, and even if detected early, there is no preventative treatment. Imaging modalities such as MRI, PET, and SPECT have the potential to contribute to both the diagnosis of Alzheimer's disease, as well as assist in the search for more effective treatments. A number of AD-related biomarkers have been proposed and evaluated. The use of PET imaging to detect alterations in regional brain metabolism using [(18)F]FDG has enabled more sensitive and accurate early diagnosis of AD, especially in conjunction with traditional medical evaluation. Additionally, magnetic resonance imaging and spectroscopy provide a wide range of biomarkers that have been shown to correlate with the progression of AD. Some of these markers have been pursued in clinical trials. Progress has been made toward the evaluation of other more AD-specific biomarkers. However, many questions remain concerning the validity and sensitivity of these imaging biomarkers, to aid in the assessment of potential new treatments, especially those related to increased levels of amyloid peptides in the brain. .COPYRGT. 2006 Bentham Science Publishers Ltd.

CT Medical Descriptors:
*Alzheimer disease: DI, diagnosis
binding affinity
brain blood flow
brain metabolism
brain region
cell activation
clinical trial
contrast enhancement
correlation analysis
diagnostic accuracy
diagnostic value
diffusion weighted imaging
disease course
drug structure
drug uptake
early diagnosis
human
image quality
imaging system
medical assessment
microglia

mouse
neurofibrillary tangle
nonhuman
*nuclear magnetic resonance imaging
nuclear magnetic resonance spectroscopy
*positron emission tomography
rat
review
senile plaque
sensitivity and specificity
*single photon emission computer tomography
validity

CT Drug Descriptors:
3 (2 azetidinylmethoxy)pyridine
4 aminobutyric acid: EC, endogenous compound
acetylcholinesterase: EC, endogenous compound
acridine orange: AN, drug analysis
amyloid beta protein: EC, endogenous compound
benzene derivative: AN, drug analysis
benzoxazole derivative: AN, drug analysis
biological marker: EC, endogenous compound
choline: EC, endogenous compound
congo red: AN, drug analysis
creatine: EC, endogenous compound
creatine phosphate: EC, endogenous compound
flavone derivative: AN, drug analysis
fluorodeoxyglucose f 18: AN, drug analysis
gadolinium
gadolinium pentetate
glutamic acid: EC, endogenous compound
glutamine: EC, endogenous compound
glycine: EC, endogenous compound
hexamethylpropylene amine oxime technetium tc 99m: AN, drug analysis
inositol: EC, endogenous compound
lactic acid: EC, endogenous compound
lipid: EC, endogenous compound
n acetylaspartic acid: EC, endogenous compound
n sec butyl 1 (2 chlorophenyl) n methyl 3 isoquinolinecarboxamide
phosphorylcholine: EC, endogenous compound
putrescine
pyridine derivative: AN, drug analysis
thioflavine: AN, drug analysis
unindexed drug

RN (3 (2 azetidinylmethoxy)pyridine) 161416-98-4; (4 aminobutyric acid) 28805-76-7, 56-12-2; (acetylcholinesterase) 9000-81-1; (acridine orange) 494-38-2, 65-61-2; (amyloid beta protein) 109770-29-8; (choline) 123-41-1, 13232-47-8, 1927-06-6, 4858-96-2, 62-49-7, 67-48-1; (congo red) 573-58-0, 80701-77-5; (creatine phosphate) 67-07-2; (creatine) 57-00-1; (fluorodeoxyglucose f 18) 63503-12-8; (gadolinium pentetate) 80529-93-7; (gadolinium) 7440-54-2; (glutamic acid) 11070-68-1, 138-15-8, 56-86-0, 6899-05-4; (glutamine) 56-85-9, 6899-04-3; (glycine) 56-40-6, 6000-43-7, 6000-44-8; (inositol) 55608-27-0, 6917-35-7, 87-89-8; (lactic acid) 113-21-3, 50-21-5; (lipid) 66455-18-3; (n acetylaspartic acid) 22304-28-5, 997-55-7; (n sec butyl 1 (2 chlorophenyl) n methyl 3 isoquinolinecarboxamide) 85532-75-8; (phosphorylcholine) 107-73-3; (putrescine) 110-60-1, 333-93-7; (thioflavine) 2390-54-7

L92 ANSWER 29 OF 36 EMBASE

ACCESSION NUMBER: 2001398041 EMBASE Full-text
TITLE: Opposite effects of low and high doses of arginine on glutamate-induced nitric oxide formation in rat substantia nigra.
AUTHOR: Castellano M.A.; Rojas-Diaz D.; Martin F.; Quintero M.; Alonso J.; Navarro E.; Gonzalez-Mora J.L.
CORPORATE SOURCE: M.A. Castellano, Facultad de Psicologia, Campus de Guajara, Universidad de La Laguna, Tenerife -38205, Canary Islands, Spain. mcastel@ull.es
SOURCE: Neuroscience Letters, (16 Nov 2001) Vol. 314, No. 3, pp. 127-130.
Refs: 19
ISSN: 0304-3940 CODEN: NELED5
PUBLISHER IDENT.: S 0304-3940(01)02295-9
COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 26 Nov 2001
Last Updated on STN: 26 Nov 2001

AB L-arginine is a very versatile amino acid that is involved in many important physiological processes such as protein, nitric oxide (NO), agmatine, putrescine, urea, L-ornithine or creatine synthesis and is essential for posttranslational arginylation of protein. The present study was designed to evaluate in vivo the effect of L-arginine on NO production in substantia nigra. In vivo spectroscopic and voltammetric studies were addressed in rats to record modifications in methemoglobin and NO levels under glutamate stimulation. Results showed that, under physiological L-arginine extracellular concentration, the intranigral infusion of glutamate produced an increase in NO levels. When a low dose of L-arginine was co-infused with glutamate, a persistent and higher increase in NO levels was observed. The co-infusion of glutamate with a moderate dose of L-arginine induced drastic and persistent NO production. It was also observed that high doses of either L-arginine or D-arginine inhibit NO production. Subsequently, these data show that L-arginine and D-arginine are involved in a mechanism that inhibits NO production. .COPYRGT. 2001 Elsevier Science Ireland Ltd. All rights reserved.

SO Neuroscience Letters, (16 Nov 2001) Vol. 314, No. 3, pp. 127-130.
Refs: 19
ISSN: 0304-3940 CODEN: NELED5
AB L-arginine is a very versatile amino acid that is involved in many important physiological processes such as protein, nitric oxide (NO), agmatine, putrescine, urea, L-ornithine or creatine synthesis and is essential for posttranslational arginylation of protein. The present study was designed to evaluate in vivo the effect of L-arginine on NO production in substantia nigra. In vivo spectroscopic and voltammetric studies were addressed in rats to record modifications in methemoglobin and NO levels under glutamate stimulation. Results showed that, under physiological L-arginine extracellular concentration, the intranigral infusion of glutamate produced an increase in NO levels. When a low dose of L-arginine was co-infused with glutamate, a persistent and higher increase in NO levels was observed. The co-infusion of glutamate with a moderate dose of L-arginine induced drastic and persistent NO production. It was also observed that high doses of either L-arginine or D-arginine inhibit NO production. Subsequently, these data show that L-arginine and D-arginine are involved in a mechanism that inhibits NO production. .COPYRGT. 2001 Elsevier Science Ireland Ltd. All rights reserved.

CT Medical Descriptors:

animal experiment
article
brain level
controlled study
dose response
drug effect
in vivo study
infusion
male
nonhuman
potentiometry
priority journal
rat
spectroscopy
stimulation
substantia nigra
synthesis

CT Drug Descriptors:
agmatine: EC, endogenous compound
amino acid: DO, drug dose
amino acid: CE, intracerebral drug administration
amino acid: PD, pharmacology
*arginine: DO, drug dose
*arginine: CE, intracerebral drug administration
*arginine: PD, pharmacology
creatinine: EC, endogenous compound
*dextro arginine: DO, drug dose
*dextro arginine: CE, intracerebral drug administration
*dextro arginine: PD, pharmacology
*glutamic acid
methemoglobin: EC, endogenous compound
*nitric oxide: EC, endogenous compound
ornithine: EC, endogenous compound
protein: EC, endogenous compound
putrescine: EC, endogenous compound
urea: EC, endogenous compound

RN (agmatine) 306-60-5; (amino acid) 65072-01-7; (arginine) 1119-34-2, 15595-35-4, 7004-12-8, 74-79-3; (creatinine) 19230-81-0, 60-27-5; (glutamic acid) 11070-68-1, 138-15-8, 56-86-0, 6899-05-4; (nitric oxide) 10102-43-9; (ornithine) 70-26-8, 7006-33-9; (protein) 67254-75-5; (putrescine) 110-60-1, 333-93-7; (urea) 57-13-6

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ACCESSION NUMBER: 2000389391 EMBASE Full-text
TITLE: Attenuation of isoproterenol-mediated myocardial injury in rat by an inhibitor of polyamine synthesis.
AUTHOR: Tipnis U.R.; He G.Y.; Li S.; Campbell G.; Boor P.J.
CORPORATE SOURCE: Dr. P.J. Boor, Department of Pathology, University of Texas, Medical Branch, Galveston, TX 77555-0609, United States. pboor@utmb.edu
SOURCE: Cardiovascular Pathology, (2000) Vol. 9, No. 5, pp. 273-280.
Refs: 60
ISSN: 1054-8807 CODEN: CATHE8
PUBLISHER IDENT.: S 1054-8807(00)00038-7
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery

030 Clinical and Experimental Pharmacology
037 Drug Literature Index
005 General Pathology and Pathological Anatomy
006 Internal Medicine

LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 13 Dec 2000
Last Updated on STN: 13 Dec 2000

AB Objective: Ornithine decarboxylase (ODC) is an initial rate-limiting enzyme in the synthesis of polyamines (putrescine, spermidine, and spermine) that play a role in cell growth and differentiation. Recent studies have shown that spermidine and spermine cause injury to a variety of cells including myocytes in vitro. In this investigation, we used α-difluoromethylornithine (DFMO), a specific and irreversible inhibitor of ODC activity and polyamine synthesis to test the hypothesis that polyamines contribute to myocardial injury in rat. Methods: Male Sprague Dawley rats were treated with (i) saline (0.2 ml/day, s.c.), (ii) isoproterenol (ISO) (5 mg/kg/day for 8 days, s.c.) to produce necrotizing myocardial injury, or with (iii) DFMO + ISO. DFMO was started 2 days before the initiation of ISO and both ISO and DFMO were continued until the end of the experimental period. Myocardial injury was assessed by determining the increased release of creatine phosphokinase (CPK) and lactate dehydrogenase (LDH) into the plasma, and by morphometric analysis of the lesion area in heart sections stained with Gomori trichrome. Results: ISO induced the release of CPK and LDH by 6 hr and 24 hr, respectively, and produced subendocardial necrosis, which was both acute and resolving following 8 days of ISO. DFMO treatment inhibited ISO-induced increases in (i) ODC activity and putrescine and spermidine levels in heart, (ii) CPK and LDH activity in plasma, and (iii) the area of subendocardial lesions. Conclusions: These observations suggest that polyamines are one of the intracellular factors that contribute to ISO-mediated cardiac injury in the rat. (C) 2000 by Elsevier Science Inc.

SO Cardiovascular Pathology, (2000) Vol. 9, No. 5, pp. 273-280.
Refs: 60
ISSN: 1054-8807 CODEN: CATHE8

AB Objective: Ornithine decarboxylase (ODC) is an initial rate-limiting enzyme in the synthesis of polyamines (putrescine, spermidine, and spermine) that play a role in cell growth and differentiation. Recent studies have shown that spermidine and spermine cause injury to a variety of cells including myocytes in vitro. In this investigation, we used α-difluoromethylornithine (DFMO), a specific and irreversible inhibitor of ODC activity and polyamine synthesis to test the hypothesis that polyamines contribute to myocardial injury in rat. Methods: Male Sprague Dawley rats were treated with (i) saline (0.2 ml/day, s.c.), (ii) isoproterenol (ISO) (5 mg/kg/day for 8 days, s.c.) to produce necrotizing myocardial injury, or with (iii) DFMO + ISO. DFMO was started 2 days before the initiation of ISO and both ISO and DFMO were continued until the end of the experimental period. Myocardial injury was assessed by determining the increased release of creatine phosphokinase (CPK) and lactate dehydrogenase (LDH) into the plasma, and by morphometric analysis of the lesion area in heart sections stained with Gomori trichrome. Results: ISO induced the release of CPK and LDH by 6 hr and 24 hr, respectively, and produced subendocardial necrosis, which was both acute and resolving following 8 days of ISO. DFMO treatment inhibited ISO-induced increases in (i) ODC activity and putrescine and spermidine levels in heart, (ii) CPK and LDH activity in plasma, and (iii) the area of subendocardial lesions. Conclusions: These observations suggest that polyamines are one of the intracellular factors that contribute to ISO-mediated cardiac injury in the rat. (C) 2000 by Elsevier Science Inc.

CT Medical Descriptors:
acute disease
animal experiment

animal model
animal tissue
article
controlled study
creatine kinase blood level
diagnostic approach route
drug effect
drug screening
enzyme activity
*heart muscle injury: DI, diagnosis
*heart muscle injury: DT, drug therapy
*heart muscle injury: ET, etiology
histopathology
lactate dehydrogenase blood level
male
muscle necrosis
nonhuman
pathophysiology
polyamine synthesis
priority journal
rat
subendothelium
tissue injury
tissue level
CT Drug Descriptors:
creatine kinase: EC, endogenous compound
*eflornithine: DV, drug development
*eflornithine: DT, drug therapy
*eflornithine: PD, pharmacology
*isoprenaline: TO, drug toxicity
*isoprenaline: PD, pharmacology
lactate dehydrogenase: EC, endogenous compound
ornithine decarboxylase: EC, endogenous compound
*ornithine decarboxylase inhibitor: DV, drug development
*ornithine decarboxylase inhibitor: DT, drug therapy
*ornithine decarboxylase inhibitor: PD, pharmacology
*putrescine: EC, endogenous compound
sodium chloride
*spermidine: EC, endogenous compound
*spermine: EC, endogenous compound
RN (creatine kinase) 9001-15-4; (eflornithine) 67037-37-0, 70052-12-9; (isoprenaline) 299-95-6, 51-30-9, 6700-39-6, 7683-59-2; (lactate dehydrogenase) 9001-60-9; (ornithine decarboxylase) 9024-60-6; (putrescine) 110-60-1, 333-93-7; (sodium chloride) 7647-14-5; (spermidine) 124-20-9, 334-50-9; (spermine) 306-67-2, 71-44-3

L92 ANSWER 31 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1995082089 EMBASE Full-text
TITLE: Serotonin uptake and its modulation in rat jejunal enterocyte preparation.
AUTHOR: Takayanagi S.; Hanai H.; Kumagai J.; Kaneko E.
CORPORATE SOURCE: S. Takayanagi, First Department of Medicine, Hamamatsu Univ. School of Medicine, 3600 Handa-cho, Hamamatsu 431-31, Japan
SOURCE: Journal of Pharmacology and Experimental Therapeutics, (1995) Vol. 272, No. 3, pp. 1151-1159.
ISSN: 0022-3565 CODEN: JPETAB
COUNTRY: United States

DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 030 Clinical and Experimental Pharmacology
037 Drug Literature Index
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 12 Apr 1995
Last Updated on STN: 12 Apr 1995

AB In order to determine the properties of the intestinal serotonin (5-HT) transport system, we studied 5-HT uptake by villous cell preparations isolated from the rat jejunum. These enterocytes, probably heterogenous in cell population, accumulated 5-HT against concentration gradient up to a concentration 28 times that in the medium; this accumulation occurred in a temperature-dependent fashion. Most of the uptake was the result of a saturable process at low substrate concentrations. The saturable uptake was inhibited by 1 mM potassium cyanide or 1 mM ouabain or by substitution of other cations and sugars for Na(+) in the medium. On the other hand, neither several amino acids nor putrescine had any effect on 5-HT uptake. Kinetic analysis yielded a K(m) value of 3.63 x 10(-7) M and a V(max) value of 5.76 pmol .ovrhdot. 10(6) cells(-1) .ovrhdot. min(-1) for this uptake process. Imipramine inhibited 5-HT uptake in a concentration-dependent fashion, with a K(i) value of 3.66 x 10(-7) M. 5-HT uptake was also inhibited by ethyleneglycol-0,0'- bis(2-aminoethyl)-N,N,N',N'-tetraacetic acid, by verapamil, by N-(6- aminohexyl)-5-chloro-1-naphthalenesulfonamide and by phorbol 12-myristate 13- acetate. These findings suggest that the present enterocyte preparation had a relatively specific secondary active transport system for 5-HT.

TI Serotonin uptake and its modulation in rat jejunal enterocyte preparation.

SO Journal of Pharmacology and Experimental Therapeutics, (1995) Vol. 272, No. 3, pp. 1151-1159.
ISSN: 0022-3565 CODEN: JPETAB

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CT Medical Descriptors:
active transport
animal cell
animal tissue
article
cell transport
concentration response
controlled study
*intestine absorption

intestine cell
intestine contraction
intestine villus
jejunum mucosa
male
nonhuman
priority journal
rat
*serotonin release
temperature dependence
transport kinetics
CT Drug Descriptors:
2 amino 2 methylpropionic acid: PD, pharmacology
asparagine: PD, pharmacology
bucladesine: PD, pharmacology
*egtazic acid: PD, pharmacology
histidine: PD, pharmacology
*imipramine: PD, pharmacology
leucine: PD, pharmacology
n (6 aminohexyl) 5 chloro 1 naphthalenesulfonamide: PD, pharmacology
*ouabain: PD, pharmacology
phorbol 13 acetate 12 myristate: PD, pharmacology
*potassium cyanide: PD, pharmacology
putrescine: PD, pharmacology
*serotonin creatinine sulfate: CR, drug concentration
*serotonin creatinine sulfate: PK, pharmacokinetics
*serotonin creatinine sulfate: PD, pharmacology
tryptophan: PD, pharmacology
verapamil: PD, pharmacology
RN (2 amino 2 methylpropionic acid) 62-57-7; (asparagine) 70-47-3, 7006-34-0; (bucladesine) 16980-89-5, 362-74-3; (egtazic acid) 67-42-5; (histidine) 645-35-2, 7006-35-1, 71-00-1; (imipramine) 113-52-0, 50-49-7; (leucine) 61-90-5, 7005-03-0; (n (6 aminohexyl) 5 chloro 1 naphthalenesulfonamide) 65595-90-6; (ouabain) 11018-89-6, 630-60-4; (phorbol 13 acetate 12 myristate) 16561-29-8; (potassium cyanide) 151-50-8; (putrescine) 110-60-1, 333-93-7; (serotonin creatinine sulfate) 971-74-4; (tryptophan) 6912-86-3, 73-22-3; (verapamil) 152-11-4, 52-53-9

L92 ANSWER 32 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1995128467 EMBASE Full-text
TITLE: Effects of polyamines on reperfusion myocardial injury after ischemia.
AUTHOR: Namiki A.
CORPORATE SOURCE: A. Namiki, Department of Internal Medicine, Jikei University School of Medicine, Tokyo, Japan
SOURCE: Tokyo Jikeikai Medical Journal, (1995) Vol. 110, No. 1, pp. 95-102.
ISSN: 0375-9172 CODEN: TJIDAH
COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
029 Clinical and Experimental Biochemistry
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: Japanese
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 23 May 1995

Last Updated on STN: 23 May 1995

AB Polyamines (Putrescine, Spermidine, Spermine) play an important role in the activation of protein and nucleic acid synthesis, which are required for cellular hypertrophy. Recently increasing attention has been placed on possible involvements of polyamines in the regulation of cardiac functions. It is thought that polyamines interact with the negatively charged phospholipid biomembranes, protecting membranes from lipid peroxidation and stabilizing membrane functions. I investigated the possible effect of polyamines to protect myocardium from reperfusion injury in isolated perfused rat hearts. The hearts from male Wistar rats (280 320 g) were perfused by the Langendorff technique. Global ischemia was continued for 20 min followed by 30 min reperfusion. The hearts were immersed in 37°C buffer during ischemia. Spermine (SPM, 200 μM) or Spermidine (SPD, 200 μM) was administered before global ischemia for 5 min. Reperfusion injury after ischemia was assessed by measuring the release of three enzymes in the coronary effluent, namely creatine phosphokinase, glutamic oxaloacetic transaminase and lactate dehydrogenase. Thereafter, the left ventricular pressure (LVP), LVdp/dt, incidence of reperfusion induced ventricular arrhythmias (VT, VF) and recovery times for sinus rhythm after reperfusion were measured. In the SPM treated group, release of three enzymes in the coronary effluent were significantly less than the non-treated control value. In the SPD treated group, release of three enzymes were not significantly less than the non-treated control value. The incidence of reperfusion induced ventricular arrhythmias in the non-treated control group, SPM treated group and SPD treated group was 36%, 0% and 14%, respectively. The recovery times for sinus rhythm in the SPM treated group (4 ±2 min) and SPD treated group (5 ±3 min), were also significantly reduced when compared with the non-treated control group (11 ± 3 min). The application of SPM or SPD caused the LVP to decrease temporarily but to increase rapidly during reperfusion. These results suggested that SPM and SPD protected myocardial membrane from reperfusion injury after ischemia even though it was low in concentrations. It is proposed that polyamines on cellular membrane properties stabilized the membrane against lipid peroxidation and anomalous calcium influx of reperfusion.

SO Tokyo Jikeikai Medical Journal, (1995) Vol. 110, No. 1, pp. 95-102.
ISSN: 0375-9172 CODEN: TJIDAH

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reduced when compared with the non-treated control group (11 ± 3 min). The application of SPM or SPD caused the LVP to decrease temporarily but to increase rapidly during reperfusion. These results suggested that SPM and SPD protected myocardial membrane from reperfusion injury after ischemia even though it was low in concentrations. It is proposed that polyamines on cellular membrane properties stabilized the membrane against lipid peroxidation and anomalous calcium influx of reperfusion.

CT Medical Descriptors:
animal tissue
article
controlled study
*heart muscle ischemia
isolated heart
lipid peroxidation
male
membrane stabilization
nonhuman
rat
*reperfusion injury: PC, prevention

CT Drug Descriptors:
aspartate aminotransferase: EC, endogenous compound
creatine kinase: EC, endogenous compound
lactate dehydrogenase: EC, endogenous compound
*polyamine: PD, pharmacology
protective agent: PD, pharmacology
putrescine: PD, pharmacology
spermidine: PD, pharmacology
spermine: PD, pharmacology

RN (aspartate aminotransferase) 9000-97-9; (creatine kinase) 9001-15-4; (lactate dehydrogenase) 9001-60-9; (putrescine) 110-60-1, 333-93-7; (spermidine) 124-20-9, 334-50-9; (spermine) 306-67-2, 71-44-3

L92 ANSWER 33 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1984179399 EMBASE Full-text
TITLE: The role of polyamines in somatomedin-stimulated differentiation of L6 myoblasts.
AUTHOR: Ewton D.Z.; Erwin B.G.; Pegg A.E.; Florini J.R.
CORPORATE SOURCE: Biology Department, Syracuse University, Syracuse, NY 13210, United States
SOURCE: Journal of Cellular Physiology, (1984) Vol. 120, No. 3, pp. 263-270.
ISSN: 0021-9541 CODEN: JCLLAX
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
029 Clinical and Experimental Biochemistry
003 Endocrinology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 1991
Last Updated on STN: 10 Dec 1991

AB The somatomedins are potent stimulators of proliferation and differentiation of cultured myoblasts. In studies on the mechanism(s) of these actions, we have measured the activities of ornithine decarboxylase (ODC), an enzyme associated with rapid cell proliferation, and creatine kinase (CK), a biochemical marker for muscle differentiation, after treatment of L6 myoblast cultures with Multiplication Stimulating Activity (MSA), a member of the

somatomedin family of insulinlike growth factors. ODC levels reached a peak 24 hours after MSA addition (before any detectable differentiation of the myoblasts) and then decreased as differentiation commenced and CK activity increased. Addition of alpha-difluoromethylornithine (DFMO), an irreversible inhibitor of ODC, caused a dramatic decrease in differentiation. Measurement of (3)H-thymidine incorporation, DNA content, and cell number established that the effect of DFMO on differentiation was not a simple consequence of its antiproliferative actions. Cellular levels of putrescine and spermidine (but not spermine) decreased substantially following addition of DFMO to the cultures. The inhibitory effects of DFMO were abolished upon addition of exogenous polyamines to the medium. However, addition of polyamines in the absence of MSA or DFMO did not mimic the stimulation of differentiation by MSA. We conclude that polyamines play an essential role in the stimulation of L6 myoblast differentiation by somatomedins, but they are not sufficient to effect this stimulation.

SO Journal of Cellular Physiology, (1984) Vol. 120, No. 3, pp. 263-270.
ISSN: 0021-9541 CODEN: JCLLAX

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CT Medical Descriptors:
article
*cell differentiation
*drug comparison
*drug efficacy
*drug mechanism
*drug metabolism
in vitro study
muscle
*myoblast
nonhuman
rat

CT Drug Descriptors:
*creatine kinase
*eflornithine
*ornithine c 14
*ornithine decarboxylase
*polyamine
putrescine
radioisotope
*somatomedin
spermidine

spermine
thymidine h 3
unclassified drug
RN (creatine kinase) 9001-15-4; (eflornithine) 67037-37-0, 70052-12-9; (ornithine decarboxylase) 9024-60-6; (putrescine) 110-60-1, 333-93-7; (spermidine) 124-20-9, 334-50-9; (spermine) 306-67-2, 71-44-3; (thymidine h 3) 50-88-4

L92 ANSWER 34 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1984066416 EMBASE Full-text
TITLE: Gustatory responses of the rainbow trout (Salmo gairdneri) palate to amino acids and derivatives.
AUTHOR: Marui T.; Evans R.E.; Zielinski B.; Hara T.J.
CORPORATE SOURCE: Department of Fisheries and Oceans, Freshwater Institute, Winnipeg, Man. R3T 2N6, Canada
SOURCE: Journal of Comparative Physiology - A Sensory, Neural, and Behavioral Physiology, (1983) Vol. 153, No. 4, pp. 423-433.
CODEN: JCPADN
COUNTRY: Germany
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 1991
Last Updated on STN: 10 Dec 1991
TI Gustatory responses of the rainbow trout (Salmo gairdneri) palate to amino acids and derivatives.
SO Journal of Comparative Physiology - A Sensory, Neural, and Behavioral Physiology, (1983) Vol. 153, No. 4, pp. 423-433.
CODEN: JCPADN
CT Medical Descriptors:
animal cell
animal experiment
*chemoreceptor
*dose response
drug administration
*drug comparison
*drug efficacy
drug response
electron microscopy
etiology
fish
gustatory system
methodology
nonhuman
*taste
*ultrastructure
CT Drug Descriptors:
*2 amino 3 guanidinopropionic acid
*2 amino 4 guanidinobutyric acid
*amino acid
*arginine
*betaine
unclassified drug
RN (amino acid) 65072-01-7; (arginine) 1119-34-2, 15595-35-4, 7004-12-8, 74-79-3; (betaine) 107-43-7, 590-46-5

L92 ANSWER 35 OF 36 DRUGU COPYRIGHT 2007 THE THOMSON CORP on STN
ACCESSION NUMBER: 1998-35099 DRUGU P N V Full-text
TITLE: Thiamine transport in human placental brush border membrane

vesicles.
AUTHOR: Grassl S M
CORPORATE SOURCE: Univ.New-York-State
LOCATION: Syracuse, N.Y., USA
SOURCE: Biochim.Biophys.Acta M (1371, No. 2, 213-22, 1998) 7 Fig. 5 Tab. 28 Ref.
CODEN: BBBMBS ISSN: 0005-2736
AVAIL. OF DOC.: Department of Pharmacology, SUNY Health Science Center, 766 Irving Avenue, Syracuse, NY 13210, U.S.A.
LANGUAGE: English
DOCUMENT TYPE: Journal
FIELD AVAIL.: AB; LA; CT
FILE SEGMENT: Literature
AB The transport of thiamine (Sigma-Chemical) across human placental brush border membrane vesicles was investigated. The amine at position 4 of the pyrimidine ring was an important determinant for interaction with the transporter substrate binding site(s). There appeared to be 3 separate organic cation exchange mechanisms mediating the transport of thiamine, guanidine (Sigma-Chemical) and methylisobutylamiloride (MIA, Research-Biochem.). Valinomycin, FCCP, cimetidine, clonidine, amiloride, choline, methylnicotinamide-N+, tetrylammonium Br, creatinine, 5-HT, histamine, dopamine, putrescine, spermidine, spermine, adenine, cytosine, pyrthiamine, amprolium, oxythiamine, thiamine-monophosphate, cocarboxylase (all Sigma-Chemical), imipramine (Research-Biochem.), harmaline (Aldrich) and pantothenate were used.
PY 1998
AB The transport of thiamine (Sigma-Chemical) across human placental brush border membrane vesicles was investigated. The amine at position 4 of the pyrimidine ring was an important determinant for interaction with the transporter substrate binding site(s). There appeared to be 3 separate organic cation exchange mechanisms mediating the transport of thiamine, guanidine (Sigma-Chemical) and methylisobutylamiloride (MIA, Research-Biochem.). Valinomycin, FCCP, cimetidine, clonidine, amiloride, choline, methylnicotinamide-N+, tetrylammonium Br, creatinine, 5-HT, histamine, dopamine, putrescine, spermidine, spermine, adenine, cytosine, pyrthiamine, amprolium, oxythiamine, thiamine-monophosphate, cocarboxylase (all Sigma-Chemical), imipramine (Research-Biochem.), harmaline (Aldrich) and pantothenate were used.
ABEX The magnitude of thiamine (1 uM) influx across human placental brush border membrane vesicles was unaffected by the imposition of an inwardly-directed Na gradient. Intravesicular thiamine accumulation was indistinguishable when measured in the presence and absence of conditions favoring the development of an inside-negative, potassium diffusion potential. The imposition of an inside-acid pH gradient (pH 6.5/5) induced accumulation of thiamine to levels exceeding equilibrium,Protonophore-induced dissipation of an imposed inside-acid pH gradient in the absence of a membrane potential abolished the accumulation of thiamine. The rate and magnitude of intravesicular 3H-thiamine accumulation was increased when measured in the presence, rather than the absence, of an outwardly directed thiamine concentration gradient. Substrate specificity studies of the proton/thiamine exchange mechanism suggested that the amine at position 4 of the pyrimidine ring, but not the hydroxyethyl side chain or an unmodified thiazolium ring is an important chemical determinant for interaction with the transported substrate binding site(s). These studies further suggested the possible presence of 3 separate organic cation exchange mechanism mediating the transport of thiamine, guanidine and MIA across the placental brush border membrane. (E61/MB)
CT [01] THIAMINE *PH; SIGMA-CHEM. *FT; GUANIDINE *RC; METHYLISOBUTYLAMILORIDE *RC; VALINOMYCIN *RC; FCCP *RC; CIMETIDINE

*RC; CLONIDINE *RC; AMILORIDE *RC; CHOLINE *RC; METHYLNICOTINAMIDE-N+ *RC; TETRYLAMMONIUM *RC; CREATININE *RC; SEROTONIN *RC; HISTAMINE *RC; DOPAMINE *RC; PUTRESCINE *RC; SPERMIDINE *RC; SPERMINE *RC; ADENINE *RC; CYTOSINE *RC; PYRTHIAMINE *RC; AMPROLIUM *RC; OXYTHIAMINE *RC; MONOPHOSPHOTHIAMINE *RC; COCARBOXYLASE *RC; IMIPRAMINE *RC; HARMALINE *RC; PANTOTHENATE *RC; THIAMINE *RN; IN-VITRO *FT; PLACENTA *FT; VITAMIN-METAB. *FT; TRANSPORT *FT; HUMAN *FT; BRUSH-BORDER *FT; MEMBRANE *FT; FLUX *FT; PH-PK *FT; SUBCELL.STRUCT. *FT; VITAMINS-B1 *FT; PH *FT

L92 ANSWER 36 OF 36 DRUGU COPYRIGHT 2007 THE THOMSON CORP on STN
ACCESSION NUMBER: 1988-36105 DRUGU B P Full-text
TITLE: 5-Fluoromethyl- orinithine, an Irreversible and Specific Inhibitor of L-ornithine: 2-oxo-acid Aminotransferase .
AUTHOR: Daune G; Gerhart F; Seiler N
CORPORATE SOURCE: Merrel-Dow
LOCATION: Strasbourg, France
SOURCE: Biochem.J. (253, No. 2, 481-88, 1988) 6 Fig. 2 Tab. 42 Ref.
CODEN: BIJOAK ISSN: 0264-6021
AVAIL. OF DOC.: Merrell Dow Research Institute, 16 rue d'Ankara, 67084, Strasbourg Cedex, France.
LANGUAGE: English
DOCUMENT TYPE: Journal
FIELD AVAIL.: AB; LA; CT; MPC
FILE SEGMENT: Literature

AB 5-fluoromethyl- orinithine MDL-72912 (5-FMOrn) specifically and irreversibly inhibited rat liver L-ornithine:2-oxoacid aminotransferase (OAT) and competitively inhibited rat prostate ornithine decarboxylase (ODC) but had no effect on rat brain 4- aminobutyrate: 2-oxoglutarate aminotransferase (GABA-T) in vitro; 5-FMOrn i.p. in mice reduced OAT and increased L-ornithine (Orn) in brain, eye and liver, while chronic 5-FMOrn increased brain Orn and putrescine and reduced carnosine and homocarnosine, and increased urinary Orn and putrescine, but had no behavioral effect. It is concluded that 5-FMOrn is the 1st specific OAT inactivator, and may be useful in the elucidation of pathological and phsiological Orn transamination, which appears to be an important but not vital pathway.

TI 5-Fluoromethyl- orinithine, an Irreversible and Specific Inhibitor of L-ornithine: 2-oxo-acid Aminotransferase.

PY 1988

AB 5-fluoromethyl- orinithine MDL-72912 (5-FMOrn) specifically and irreversibly inhibited rat liver L-ornithine:2-oxoacid aminotransferase (OAT) and competitively inhibited rat prostate ornithine decarboxylase (ODC) but had no effect on rat brain 4- aminobutyrate: 2-oxoglutarate aminotransferase (GABA-T) in vitro; 5-FMOrn i.p. in mice reduced OAT and increased L-ornithine (Orn) in brain, eye and liver, while chronic 5-FMOrn increased brain Orn and putrescine and reduced carnosine and homocarnosine, and increased urinary Orn and putrescine, but had no behavioral effect. It is concluded that 5-FMOrn is the 1st specific OAT inactivator, and may be useful in the elucidation of pathological and phsiological Orn transamination, which appears to be an important but not vital pathway.

ABEX OAT prepared from livers of male Sprague-Dawley rats was irreversibly inactivated by 5-FMOrn with pseudo-first-order kinetics, apparent Ki 70 uM and half life at infinite inhibitor concentration of 1.1 min. 5-FMOrn competitively reduced the rate of Orn decarboxylation but not carbamoylation, with Ki 0.3 mM. 5-FMOrn was a poor substrate of ODC. 5-FMOrn 10 mg/kg i.p. in female CD1 mice rapidly reduced OAT activity to a minimum of 10-20% of total activity, and increased Orn concentration in brain, eye and liver after 2-24 hr, but Orn concentrations started to fall again before significant recovery of OAT activity. Subsequent in

vitro incubation with 5-FMOrn of liver and brain homogenates from mice treated in vivo failed to augment the OAT inhibition. Liver and eye putrescine concentrations were unaffected by 5-FMOrn 10 mg/kg/day i.p. x 14 days, residual OAT activity was 21% in brain 23%, in liver and 27% in eye; Orn was significantly increased in brain and eye, and putrescine was increased and carnosine and homocarnosine reduced in brain. In 2 mice given chronic 5-FMOrn, urinary Orn and putrescine were increased but urea and creatinine unaltered. No behavioral effects were seen. (W76/SJB) (N.S.).

[01] MDL-72912 *PH; TRIAL-PREP. *FT; EC-2.6.1.13 *FT; EC-4.1.1.17 *FT; EC-2.6.1.19 *FT; INHIBITION *FT; RAT *FT; LIVER *FT; IN-VITRO *FT; MITOCHONDRIA *FT; MOUSE *FT; IN-VIVO *FT; I.P. *FT; BRAIN *FT; EYE *FT; CARNOSINE *FT; HOMOCARNOSINE *FT; PUTRESCINE *FT; CONC. *FT; URINE *FT; CHRON. *FT; ACUTE *FT; NEW *FT; POLYAMINE *FT; ALKYLFLUORIDE *FT; AMINOACID *FT; N-METAB. *FT; ORNITHINE-OXO-ACID-AMINOTRANSFERASE *FT; ORNITHINE-DECARBOXYLASE *FT; AMINOBUTYRATE-AMINOTRANSFERASE *FT; LAB.ANIMAL *FT; SUBCELL.STRUCT. *FT; LAB.ANIMAL *FT; INJECTION *FT; PH *FT; MDL-72912 *RN

Full search history

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                D L1
                D SCAN

     FILE 'REGISTRY' ENTERED AT 13:56:08 ON 28 NOV 2007
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L3              1 SEA ABB=ON  PLU=ON  56-85-9/RN
L4              1 SEA ABB=ON  PLU=ON  107-43-7/RN
L5              1 SEA ABB=ON  PLU=ON  333-93-7/RN
L6              1 SEA ABB=ON  PLU=ON  353-09-3/RN
L7              1 SEA ABB=ON  PLU=ON  835598-36-2/RN
L8              1 SEA ABB=ON  PLU=ON  835598-38-4/RN
L9              1 SEA ABB=ON  PLU=ON  625-08-1/RN
L10             1 SEA ABB=ON  PLU=ON  57-00-1/RN
L11             1 SEA ABB=ON  PLU=ON  110-60-1/RN
L12             1 SEA ABB=ON  PLU=ON  107-43-7/RN

     FILE 'HCAPLUS' ENTERED AT 13:59:36 ON 28 NOV 2007
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L16           245 SEA ABB=ON  PLU=ON  L5
L17           369 SEA ABB=ON  PLU=ON  L6
L18             2 SEA ABB=ON  PLU=ON  L7
L19             1 SEA ABB=ON  PLU=ON  L8
L20           370 SEA ABB=ON  PLU=ON  L9
L21          6901 SEA ABB=ON  PLU=ON  L10
L22         13138 SEA ABB=ON  PLU=ON  L11
L23          5908 SEA ABB=ON  PLU=ON  L12
L24           790 SEA ABB=ON  PLU=ON  ((MONO?)(3A)CREATIN?)
L25            15 SEA ABB=ON  PLU=ON  DICREATIN?
L26         11670 SEA ABB=ON  PLU=ON  PUTRESCIN?
L27           125 SEA ABB=ON  PLU=ON  (GUANIDIN?(3A)PROPION?)
L28           728 SEA ABB=ON  PLU=ON  (HYDROXY?)(3A)(METHYLBUTYR?)
L29          2097 SEA ABB=ON  PLU=ON  ((L13 OR L14 OR L15 OR L16 OR L17 OR L18
                  OR L19)) AND (ENTER? OR PARENTER?)
L30         30974 SEA ABB=ON  PLU=ON  L21 OR CREATINE? OR L24 OR L25
L31         15982 SEA ABB=ON  PLU=ON  L16 OR L22 OR PUTRESCINE?
L32            20 SEA ABB=ON  PLU=ON  (PUTRESCIN?(2A)HYDROCHLOR?)
L33        146242 SEA ABB=ON  PLU=ON  L2 OR ALANINE?
L34         52786 SEA ABB=ON  PLU=ON  L3 OR GLUTAMINE
L35          6019 SEA ABB=ON  PLU=ON  L15 OR L23 OR TRIMETHYLGLYCINE OR (TRIMETHY
                  L(2A)GLYCINE)
L36           616 SEA ABB=ON  PLU=ON  L17 OR L27 OR GUANIDINOPROPION?
L37            45 SEA ABB=ON  PLU=ON  L30 AND (L20 OR L28)
L38             0 SEA ABB=ON  PLU=ON  L32 AND L33 AND L34 AND L35 AND L36
L39             8 SEA ABB=ON  PLU=ON  L30 AND L31 AND L33 AND L34
L40             2 SEA ABB=ON  PLU=ON  L39 AND L35
L41             1 SEA ABB=ON  PLU=ON  L39 AND L36
L42             0 SEA ABB=ON  PLU=ON  L32 AND L37
L43             2 SEA ABB=ON  PLU=ON  L18 OR L19
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L44           2 SEA ABB=ON  PLU=ON  L43 AND (ADMINIST? OR ENTER? OR PARENTER?
                OR SUPPLEM? OR ADDITI? OR PERFORMAN? OR SPORT?)
L45        2097 SEA ABB=ON  PLU=ON  L29 AND (ADMINIST? OR ENTER? OR PARENTER?
                OR SUPPLEM? OR ADDITI? OR PERFORMAN? OR SPORT?)
L46         928 SEA ABB=ON  PLU=ON  L29 AND (ADMINIST? OR TREAT? OR MEDICI? OR
                MEDICAT? OR DOSE? OR DOSA? OR SUPPLEM?)
L47           0 SEA ABB=ON  PLU=ON  L20 AND L21 AND L22 AND L23
L48          53 SEA ABB=ON  PLU=ON  L30 AND L31
L49          20 SEA ABB=ON  PLU=ON  L48 AND (L33 OR L34)
L50           1 SEA ABB=ON  PLU=ON  L13 AND L14 AND L15 AND L16 AND L17
L51           0 SEA ABB=ON  PLU=ON  L30 AND L32
L52           9 SEA ABB=ON  PLU=ON  ((L38 OR L39 OR L40 OR L41 OR L42 OR L43
                OR L44)) OR L47 OR L50 OR L51
L53           0 SEA ABB=ON  PLU=ON  L29 AND L32
L54           5 SEA ABB=ON  PLU=ON  L32 AND (ADMINIST? OR THERAP? OR TREAT? OR
                PERFORM? OR SPORT? OR DIET? OR ENTER? OR PARENTER?)
L55          14 SEA ABB=ON  PLU=ON  (L52 OR L53 OR L54)
L56        1915 SEA ABB=ON  PLU=ON  L35 AND (ADMINIST? OR THERAP? OR TREAT? OR
                PERFORM? OR SPORT? OR DIET? OR ENTER? OR PARENTER?)
L57         260 SEA ABB=ON  PLU=ON  L36 AND (ADMINIST? OR THERAP? OR TREAT? OR
                PERFORM? OR SPORT? OR DIET? OR ENTER? OR PARENTER?)
L58           2 SEA ABB=ON  PLU=ON  L56 AND L57
L59          15 SEA ABB=ON  PLU=ON  L55 OR L58
L60          36 SEA ABB=ON  PLU=ON  L37 AND (ADMINIST? OR THERAP? OR TREAT? OR
                PERFORM? OR SPORT? OR DIET? OR ENTER? OR PARENTER?)
L61           3 SEA ABB=ON  PLU=ON  L60 AND L33 AND L34
L62          17 SEA ABB=ON  PLU=ON  L59 OR L61
L63          45 SEA ABB=ON  PLU=ON  L37 AND L30
L64          36 SEA ABB=ON  PLU=ON  L63 AND (ADMINIST? OR THERAP? OR TREAT? OR
                PERFORM? OR SPORT? OR DIET? OR ENTER? OR PARENTER?)
L65           1 SEA ABB=ON  PLU=ON  L64 AND L31
L66        5865 SEA ABB=ON  PLU=ON  L31 AND (ADMINIST? OR THERAP? OR TREAT? OR
                PERFORM? OR SPORT? OR DIET? OR ENTER? OR PARENTER?)
L67          70 SEA ABB=ON  PLU=ON  L66 AND L16
L68           2 SEA ABB=ON  PLU=ON  L67 AND L30
L69          18 SEA ABB=ON  PLU=ON  L59 OR L61 OR L65 OR L68
L70           1 SEA ABB=ON  PLU=ON  L64 AND L66
L71          18 SEA ABB=ON  PLU=ON  L69 OR L70
L72             QUE ABB=ON  PLU=ON  AY<2004 OR PY<2004 OR PRY<2004 OR REVIEW/DT

L73          14 SEA ABB=ON  PLU=ON  L71 AND L72
                SAVE TEMP L73 BET232HCTX/A
                E BOLDT M?/AU
L74           6 SEA ABB=ON  PLU=ON  ("BOLDT M"/AU OR "BOLDT MATTHIAS"/AU)
                D L74 1-6 AU
                SAVE TEMP L74 BET232HCIN/A

     FILE 'MEDLINE, BIOSIS, EMBASE, DRUGU' ENTERED AT 14:52:16 ON 28 NOV 2007
L75          18 SEA ABB=ON  PLU=ON  L73
L76           6 SEA ABB=ON  PLU=ON  (PUTRESCIN?) AND (CREATIN? OR MONO(3N)
                CREATIN? OR DICREATIN?) AND ALANIN? AND GLUTAM?
L77           9 SEA ABB=ON  PLU=ON  (PUTRESCIN?) AND (CREATIN? OR MONO(3N)
                CREATIN? OR DICREATIN?) AND GUANIDIN?
L78          27 SEA ABB=ON  PLU=ON  (L75 OR L76 OR L77)
L79          48 SEA ABB=ON  PLU=ON  (PUTRESCIN?) AND (CREATIN? OR MONO(3N)
                CREATIN? OR DICREATIN?) AND (ENTER? OR PARENTER? OR ADMINIST?
                OR SUPPLE? OR TREAT?)
L80          69 SEA ABB=ON  PLU=ON  L78 OR L79
L81           6 SEA ABB=ON  PLU=ON  L80 AND ALANIN? AND GLUTAM?
L82          17 SEA ABB=ON  PLU=ON  L80 AND AMINO?
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L83          20 SEA ABB=ON  PLU=ON  L81 OR L82
L84          32 SEA ABB=ON  PLU=ON  L78 OR L83
L85          30 SEA ABB=ON  PLU=ON  L84 AND L72
                SAVE TEMP L85 BET232MLTX/A
L86          49 SEA ABB=ON  PLU=ON  L74
L87           0 SEA ABB=ON  PLU=ON  L86 AND (CREATIN? OR MONO(3N) CREATIN? OR
                DICREATIN?)
L88           0 SEA ABB=ON  PLU=ON  L86 AND PUTRESCIN?
L89          20 SEA ABB=ON  PLU=ON  L86 AND (ADMINIST? OR TREAT? OR SUPPLEM?
                OR SPORT? OR PERFORM? OR THERAP? OR PHARMAC?)
L90          19 SEA ABB=ON  PLU=ON  L89 AND L72
                SAVE TEMP L90 BET232MLIN/A
                D QUE L74
                D QUE L90

     FILE 'HCAPLUS, MEDLINE, BIOSIS, EMBASE, DRUGU' ENTERED AT 15:08:29 ON 28
     NOV 2007
L91          16 DUP REM L74 L90 (9 DUPLICATES REMOVED)
                      ANSWERS '1-6' FROM FILE HCAPLUS
                      ANSWERS '7-10' FROM FILE MEDLINE
                      ANSWERS '11-15' FROM FILE BIOSIS
                      ANSWER '16' FROM FILE EMBASE
                D L91 1-16 IBIB AB
                D QUE L73
                D QUE L85
L92          36 DUP REM L73 L85 (8 DUPLICATES REMOVED)
                      ANSWERS '1-14' FROM FILE HCAPLUS
                      ANSWERS '15-19' FROM FILE MEDLINE
                      ANSWERS '20-26' FROM FILE BIOSIS
                      ANSWERS '27-34' FROM FILE EMBASE
                      ANSWERS '35-36' FROM FILE DRUGU
                D L92 1-14 IBIB ED ABS HITIND
                D L92 15-36 IBIB AB HIT
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